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CLINICAL MALNUTRITION IN ITALY IN 1945*

Nutritional Status of Selected Population Groups

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THIS report describes a practical survey procedure for rapid clinical assessment of the nutritional status of selected population groups within a given community or area. It summarizes the results of thirty-five such studies performed in Italy during the winter, spring and summer of 1945. Approximately 4000 persons were examined. Data derived from 3109 children and pregnant or nursing women selected from refugee, institutionalized, school and population-at-large groups are presented.

By rapid, standardized assessment operations, carried out over a wide geographic area and utilizing a consistent weighted sampling technic, it was possible to gather epidemiologic information on malnutrition. This implies the determination of the extent, distribution, type and severity of existent clinical malnutrition, as evidenced by generally accepted diagnostic signs and supported by simple laboratory procedures. Data gathered in this manner were recorded on standardized punch cards, promptly analyzed and reported and used as the basis for recommendations that guided concurrent feeding operations.

GEOGRAPHIC LOCATION AND POPULATION GROUPS

Thirty-five individual nutrition surveys were performed in a wide geographic zone that included a portion of southern Italy, as well as the central and northern regions (Fig. 1).

Detailed studies were made in the Rome, Florence and Milan regions; each area had characteristic ethnic and socioeconomic differences — often accentuated by wartime alterations in transportation, housing and sanitation. Because of migration from the comparatively localized areas of destruction toward relatively intact cities, the main bulk of the population studied was urban, although several semirural and rural groups were investigated. In each survey attention was directed toward various stress population groups in which circumstances were weighted in favor of malnutrition.

The 4000 Italians examined were composed almost exclusively of children and pregnant or nursing

FIGURE 1. *Geographic Zones Involved in Nutrition-Survey Studies.*

The circles represent general areas in which groups of both urban and semirural surveys were centered.

women drawn from schools, from the general population and from institutions. School children were studied in Rome, Florence, Forli and Milan. In the same areas and in Vechiano, the general population was encountered principally within maternal and child-welfare stations. Refugee groups of dis-

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placed Italians were examined in the vicinity of Rome and Forli. Institutionalized populations were drawn from state and private orphan asylums, foundling homes and penal institutions of the various areas.

Methods of Selection

The purpose of the sampling procedure employed was to obtain information regarding the nutritional status of a large population group by standardized examination of only a representative sample of the total group. True random sampling of a total population was not feasible, since the required statistics either were not available or had lost their validity, but by deliberately introducing a factor of selection in addition to the factor of chance, it was possible to modify and direct subsequent results in a predetermined and specific direction in such fashion as to disclose existent malnutrition. This, in general, was accomplished in two ways. First, in accordance with the weighted sampling procedure that Meiklejohn[1] developed in his Wales surveys, pregnant or lactating women and children who were experiencing rapid growth spurts were served to indicate the aim and method of the study and to stimulate the necessary interest and cooperation. Specific routine questioning was designed to achieve subdivision of the area into smaller compartments, each roughly homogeneous from a socioeconomic standpoint. Within each homogeneous zone, foci of nutritional need were further localized among school, refugee or institutionalized populations on the basis of the best available data. The resultant information afforded quite reliable selection of six to eight representative examples of the neediest schools, institutions and clinics in divergent parts of the community.

Data pertaining to further definition of the population to be studied were obtained at each selected school or institution, and nominal lists of actual attendees, grouped by age or class, were inspected. An appropriate sample was attained by arbitrarily selecting from the lists each *N*th individual, *N* being determined in relation to actual attendance per age group. The sample size varied from 13 to 100 per cent of the sample eligibles (Table 1). The name, age, sex and place of origin of the selected subjects

Table 1. *Number of Cases Examined according to Area and Population Group.**

Area	Census Data				Cases Examined		
	Total estimated census	Actual census of selected area	Sample eligibles in selected area†	Percentage of eligibles examined	Children	Pregnant or lactating women	Total
Sante Croce and Cinecitta	4,970	3,000	1,850	13	267	73‡	340
Rome schools	112,000	6,020	4,428	25	1,109	0	1,109
Rome institutions	16,880	981	730	75	372	396‡	768
Florence schools	17,552	320	300	30	125	0	125
Florence institutions	3,751	312	312	36	104	80	184
Forli schools, institutions and refugees	8,000	1,190	256	67	172	17	189
Vechiano	1,100	1,000	600	17	89	11	100
Milan schools and institutions	110,000	2,124	1,300	23	295	0	295
Totals	274,253	14,947	9,776	32	2,533	577	3,110

*More than 1000 cases examined in association with Unitarian-Congregational Medical Nutrition Mission at Fondi and Pontecorvo are not included.

†Eligibility depended on three factors: age groups, pregnancy or lactation and actual attendance at school or institution.

‡Includes 75 to 80 per cent of pregnant or lactating women.

deliberately selected as representing physiologic stress groups likeliest to manifest clinical evidence of malnutrition. These categories were recipients of special allowances, but it seemed unlikely that the minimal dietary supplementation would have significantly altered their status. Secondly, on the basis of carefully evaluated local information from various sources, mothers and children from poorer socioeconomic areas were chosen. It was believed that such selection would provide a heavily weighted sample representative of the worst — rather than the average — nutrition in a given area. From a physiologic standpoint, it seemed probable that if these selected groups showed little evidence of malnutrition, no significant amount of malnutrition would exist in the remaining population.

In practice, the first contacts in any urban area were with a responsible Italian official and often with the local health officer. A short conference were inscribed by local personnel on standard punch cards. Each child or mother was then prepared for examination and queued at an appointed hour with the punch card in hand.

This general procedure was duplicated in dealing with semi-official and private organizations. In some, particularly in several refugee camps and in the general-population studies in rural or semirural areas, the procedure varied slightly: family or dwelling units were used as the basis for area sampling within the confines of a given camp, and a total census of pregnant and nursing mothers attending maternal and child health centers in rural or semirural areas was frequently examined. In general, careful sample selection and local preparations for examination of subjects from any one area rarely required more than twenty-four to forty-eight hours, and the examination itself, rarely more than two to four hours.

Examination Procedure and Reporting

The basic examination procedure consisted of a rapid inspection for signs of deficiency disease: the condition of the skin of the hands, arms, legs and thorax was noted, and the eyes, nose, lips, teeth, gums, tongue, ears, neck and skeletal structures were examined. Neurologic examination was limited to observation of gait, calf and nerve tenderness in the lower extremities; when these signs were positive, reflexes and vibration sense were tested. The presence of visible, palpable thyroid enlargement and of edema was noted. Data on height and weight were collected routinely. In most cases, Sahli hemoglobin determinations, by means of earlobe punctures, were carried out on alternate subjects in the sample concerned.

It was planned to perform gravimetric copper-sulfate protein determinations[2] in the field; with the exception of rare cases of infantile inanition encountered in pediatric hospitals or foundling homes, however, no subjects were observed in whom plasma-protein determinations seemed clinically indicated. Consequently, protein determinations were carried out only in the rare event that pediatric hospital laboratory facilities were functioning and accessible. No satisfactory field apparatus for measuring plasma volume was available. There is laboratory evidence that determination of plasma-protein concentration, without concomitant plasma-volume measurement, yields misleading and equivocal data, particularly in protein deficiency states.[3, 4]

Except for infants, patients were only partially disrobed. The thorax and abdomen of mothers were not examined routinely. Two physicians alternated in the performance of clinical examinations and Sahli hemoglobin determinations. A relatively high degree of similarity in technic and diagnosis was attained by both observers. Diagnoses were recorded by means of a moderately complete, systematized numerical code that involved approximately one hundred and twenty clinical signs of deficiency disease, the numbers of which had been printed on the standard survey cards.[5] After short experience, code numbers of the majority of signs were easily recalled. During the course of the examination, numbers on a given patient's card corresponding to observed signs were circled by an assistant. This method of recording observations was found to be quite efficient in minimizing friction, error and delay. Recording of hemoglobin levels in terms of fairly broad ranges contributed to ease in performance and subsequent analysis.

On completion of a survey, the card of each subject was punched in accordance with the numbers circled on it. All cards were sorted, and the data transcribed on detailed standard work sheets, analyzed and reported, usually within twenty-four to forty-eight hours after completion of the survey.

Clinical Criteria

Since various biochemical lesions of mild deficiency states cannot be readily and conclusively detected at a clinical level without associated laboratory aids,[6] the categorization of malnutrition was restricted to classic deficiency disease. There are considerable variations in the criteria used for such diagnoses, but in these studies the following were required:

Rickets. All subjects had at least three skeletal signs of rickets, including marked frontoparietal bossing with or without craniotabes, Harrison's groove, marked enlargement of costochondral junctions (so-called "beading" or "rosary"), clinically enlarged epiphyses or deformity of the long bones — the last being defined as deformity of lower extremities of such a degree that the distance between malleoli at proximal or distal ends of the tibias was 3 cm. or more. No roentgenographic or laboratory facilities for corroborative diagnosis or for determination and assessment of rachitic activity were available. Consequently all rickets in children four years of age or over was diagnosed as inactive or old; rickets in younger children was arbitrarily called active or recent on the basis of physiologic and statistical chance.[7, 8]

Ariboflavinosis. All cases so diagnosed had cheilosis, that is, angular stomatitis associated with denudation with or without fissuring of the lower lip, magenta tongue with fungiform hypertrophy and sometimes classic nasomalar seborrheic dermatosis. Circumlimbal injection was noted but was not considered in the diagnosis.

Vitamin A deficiency. All cases so categorized were observed in subjects with lackluster dryness of the conjunctivas and moderate or marked conjunctival thickening (excluding ptyergia and pingueculas), with or without Bitot's spots, as well as typical keratosis follicularis of both upper and lower extremities. Acneform and impetiginous skin lesions were noted but were not requisite for the diagnosis.

Inanition. In all such cases there were obvious loss of subcutaneous tissue, dehydration, loss of turgor with looseness and wrinkling of the skin, prominent bones and protuberant abdomen, with associated growth retardation or weight loss. The inclusive term "inanition" as applied, therefore, is synonymous with marasmus, infantile dystrophy, infantile atrophy and so forth.

Anemia. This diagnosis was arbitrarily designated when Sahli hemoglobin levels of 8 gm. per 100 cc. or less in infants under two years of age and 10 gm. per 100 cc. or less in children and in adults were observed — normal hemoglobin values for infants under two years of age are approximately 1 or 2 gm. less than those for older children and adults.[9–11] These arbitrary levels probably repre-

sented moderately severe anemia, although the range of normal variation is apparently large.[9–12] The standard for the instrument used was 14.6 to 15.6 gm. per 100 cc.

Other diseases. No lesions typical or strongly suggesting pellagra, beriberi, scurvy or hypoproteinemia were observed.* Associated or conditioning diseases were recorded when evident or when adequate data were available.

Results

Rickets was the most frequent classic deficiency disease observed. The incidence in particular surveys varied from 25 to 30 per cent among school children to 60 per cent among younger children and institutionalized infants. The overall incidence of rickets in the total sample was 27.7 per cent and proportionally equal in the groups over and under four years of age. This overall incidence is somewhat lower than that observed by Follis, Jackson, Eliot and Park[13] among children of Italian descent in their hospital-population study, although such a comparison is not entirely valid, since the method of diagnosis differed.

Although suggestive signs of ariboflavinosis were often noted among children three to twelve years of age, diagnostic clinical cases were rarely encountered, the incidence varying by surveys from 0 to 2 per cent.

Suggestive signs of vitamin A deficiency were noted occasionally; the incidence of keratosis follicularis, for example, varied by areas from 0 to 22 per cent. No keratomalacia was observed, although the regional occurrence of eye diseases ascribed by Italian physicians to vitamin A deficiency was apparently not infrequent in the prewar years.[14, 15] The general incidence of clinical vitamin A deficiency approximated 1.5 per cent.

Inanition, noted only among refugee or institutionalized infants two years of age and under, was frequently conditioned by a previous or current infectious process, and was often independent of available food supply. Approximately 3.0 to 7.0 per cent of institutionalized infants were classified as showing inanition — an incidence of 0.7 per cent in the total child population studied. Among the same groups of infants two years of age and under and in some groups of institutionalized preschool children, height and weight values were not infrequently below those for the national average; this might have been anticipated in view of the weighted sampling procedure employed. Neither inanition nor significant degrees of growth retardation were demonstrable among other groups of children studied.

With the exception of the previously mentioned data on institutionalized infants and young children, the 80 percentile range and median values for height and weight of central and northern Italian preschool and school-age children showed no marked deviations from prewar Italian standards.[16]

The incidence of moderately severe anemia varied from 3.0 to 20.0 per cent among institutionalized infants and preschool children, but was only 0.5 per cent among school children and 1.5 per cent among pregnant and lactating women. Since procedures other than hemoglobin determinations were not attempted under existing field conditions, it is quite possible that some of the cases of anemia observed were of non-nutritional origin.

Simple diffuse thyroid enlargement was noted in 5 to 9 per cent of children and in 18 to 23 per cent of pregnant and nursing women. In some samples the incidence of such enlargement in children under ten years of age approached 9 or 10 per cent. The geographic distribution of this finding was fairly uniform. No cases of hyperthyroidism or hypothyroidism and no cretins were seen. The association between thyroid enlargement and fluorosis was not investigated but was observed particularly in Campagna da Roma, where endemic dental fluorosis is almost universal among the static population.

Completely negative examinations were rare (2.0 to 4.5 per cent), and many isolated signs but no other classic deficiency diseases were observed. Gross psychologic or psychomotor alterations, such as apathy, listlessness, excessive irritability, anxiety and depression, were not particularly apparent.

Data pertaining to the incidence of deficiency signs and diagnoses are recorded in Tables 2, 3, 4, 5 and 6.

With the exception of inanition and infantile anemia there was little significant difference in the occurrence of classic deficiency disease in the various population categories of the respective age and sex groups.

Discussion

In thirty-five surveys involving widely scattered stress population groups totaling approximately 4000 subjects, it was not possible to demonstrate any gross clinical malnutrition serious enough in degree or wide enough in distribution to constitute an acute relief feeding problem. With rare exceptions, no deficiencies were encountered that could not have been prevented or successfully treated with cod-liver oil or dry skimmed milk, as well as timely and intelligent pediatric care.

In all surveys such deficiency disease as was encountered consistently followed a fairly uniform pattern indicative of mild chronic or subacute malnutrition. Minor regional differences in the severity of lesions were noted, but the basic pattern of deficiency showed relatively little variation. The mildness and uniformity of the nutritional deficiencies are not particularly surprising when general approximate food consumption, based on budgetary-analysis data gathered by competent workers (Table 7) is considered.[17–19]

*Gingivitis was not considered to be of scorbutic origin unless edema, cyanosis, retraction, loss of interdental papillae and bleeding were associated findings. Nonspecific glossitis was noted and described, but was not interpreted in relation to any specific deficiency.

TABLE 2. *Incidence of Deficiency Disease among 577 Pregnant or Lactating Women according to Population Group.*

GROUP	NO. OF WOMEN EXAMINED	NEGATIVE PHYSICAL EXAMINATION		SUGGESTIVE PHYSICAL SIGNS OF DEFICIENCY*		CLASSIC DEFICIENCY DISEASE		OLD RICKETS		ARIBOFLAVINOSIS		VITAMIN A DEFICIENCY		ANEMIA†		HEMOGLOBIN DETERMINATIONS	
		NO. OF CASES	PERCENTAGE	NO. OF CASES	PERCENTAGE	NO. OF CASES	PERCENTAGE	NO. OF CASES	PERCENTAGE	NO. OF CASES	PERCENTAGE	NO. OF CASES	PERCENTAGE	NO. OF CASES	PERCENTAGE	NO. OF CASES	PERCENTAGE
Refugee	82	0	0	63	76.8	19	23.1	0	0	4	4.9	15	18.3	0	0	0	0
Institutional	109	2	1.8	106	97.3	1	0.9	1	0.9	0	0	0	0	1	1.4	70	64.2
General population	386	8	2.0	378	98.0	0	0	0	0	0	0	0	0	3	4.3	188	49.0
Totals	577	10		547		20		1		4		15		4		258	
Averages			1.7		94.8		3.5		0.2		0.7		2.6		5.7		44.7

*Although such signs were observed, no classic deficiency disease was present.

†Diagnosis based on Sahli hemoglobin values of 10 gm. per 100 cc. or below. Determinations made in 70 cases of total population studied.

TABLE 3. *Incidence of Deficiency Disease among 2533 Children according to Age and Population Group.*

GROUP AND AGE OF CHILDREN	NO. OF CHILDREN EXAMINED	NEGATIVE PHYSICAL EXAMINATION		SUGGESTIVE PHYSICAL SIGNS OF DEFICIENCY*		CLASSIC DEFICIENCY DISEASE		RECENT RICKETS		OLD RICKETS		ARIBOFLAVINOSIS		VITAMIN A DEFICIENCY		INANITION		ANEMIA†		HEMOGLOBIN DETERMINATIONS PERFORMED	
		NO. OF CASES	PERCENTAGE	NO. OF CASES	PERCENTAGE	NO. OF CASES	PERCENTAGE	NO. OF CASES	PERCENTAGE	NO. OF CASES	PERCENTAGE	NO. OF CASES	PERCENTAGE	NO. OF CASES	PERCENTAGE	NO. OF CASES	PERCENTAGE	NO. OF CASES	PERCENTAGE	NO. OF CASES	PERCENTAGE
Refugee																					
2 years and under	71	10	14.0	37	52.1	24	33.8	22	30.9	0	0	0	0	0	0	3	4.2	0	0	0	0
3-5 years	71	1	1.4	24	33.8	46	64.8	24	33.8	15	21.1	0	0	10	14.0	0	0	0	0	0	0
6-9 years	90	0	0	53	58.9	37	41.1	0	0	27	30.0	0	0	19	21.1	0	0	0	0	0	0
10-15 years	90	2	2.2	56	62.2	32	35.5	0	0	7	7.7	0	0	29	32.2	0	0	0	0	0	0
Institutional																					
2 years and under	183	15	8.1	92	50.3	76	41.6	63	34.9	0	0	0	0	0	0	14	7.6	3	2.1	129	70.5
3-5 years	95	0	0	18	18.9	77	81.0	37	38.9	40	42.1	0	0	0	0	0	0	9	6.4	48	50.5
10-15 years	151	0	0	128	84.8	23	15.2	0	0	22	14.6	0	0	1	0.7	0	0	0	0	75	49.0
General population																					
2 years and under	63	1	1.9	27	42.8	35	52.4	35	52.4	0	0	0	0	0	0	0	0	0	0	11	17.5
3-5 years	278	10	3.6	165	59.3	103	37.1	27	9.7	74	26.6	1	0.3	1	0.3	0	0	0	0	93	33.4
6-9 years	829	40	4.8	573	69.1	216	26.0	0	0	227	27.4	11	1.3	3	0.4	0	0	3	0.8	377	45.5
10-15 years	612	35	5.7	460	75.2	117	19.1	0	0	80	13.1	15	2.4	7	1.1	0	0	0	0	209	34.2
Totals	2533	114		1633		786		208		492		27		70		17		15		942	
Averages			4.5		64.5		31.0		8.3		19.4		1.1		2.8		0.7		1.6		37.2

*Although such signs were observed, no classic deficiency disease was present.

†Diagnosis based on Sahli hemoglobin values of 8 gm. per 100 cc. or less in infants under two years of age, and values of 10 gm. per 100 cc. in children over two years of age. Determinations made in 140 institutionalized children under two years of age, in 141 institutionalized children three to five years of age and in 377 children in the general population six to nine years of age.

TABLE 4. *Deficiency Signs Observed among 404 Refugee Children and Pregnant or Lactating Women.*

GROUP	NO. OF SUBJECTS EXAMINED	NEGATIVE PHYSICAL EXAMINATION	FRONTAL BOSS	HARRISON'S GROOVE	RACHITIC ROSARY	BOW LEGS AND DEFORMITY OF LONG BONES	ENLARGED EPIPHYSIS	RACHITIC POT BELLY	NONSPECIFIC GLOSSITIS	NONSPECIFIC GINGIVITIS	DENUDED LOWER LIP	ANGULAR STOMATITIS	CHEILOSIS	KERATOSIS FOLLICULARIS	SEBORRHEIC DERMATOSIS	MODERATE CONJUNCTIVAL THICKENING	CONJUNCTIVAL DRYNESS	MAGENTA TONGUE, WITH FOLLICULAR HYPERTROPHY	BITOT'S SPOTS	CALF TENDERNESS	NERVE TENDERNESS	THYROID HYPERTROPHY	INANITION
Children																							
2 years of age and under....	71	10	54	19	24	13	24	19	2	0	1	0	0	3	2	0	0	0	0	0	0	1	3
3–5 years of age	71	1	54	31	31	46	27	42	33	8	6	14	2	25	1	0	29	2	0	3	0	1	0
6–9 years of age	90	0	46	29	37	25	29	0	41	26	10	16	4	30	2	1	52	3	1	13	3	4	0
10–15 years of age	90	2	12	10	21	22	16	0	37	35	5	21	2	43	4	11	62	7	10	19	3	11	0
Totals	322	13	166	89	113	106	96	61	113	69	22	51	8	101	9	12	143	12	11	35	3	16	3
Percentages		4.0	56.5	27.6	35.1	32.9	29.9	18.9	35.1	21.4	6.8	15.8	2.5	31.4	2.8	3.7	44.4	3.7	3.4	10.9	0.9	5.0	0.9
Pregnant or lactating women..	82	0	0	0	0	0	0	0	31	51	5	19	5	19	5	19	56	14	14	13	4	18	0
Percentages									37.8	62.0	6.1	23.1	6.1	23.1	6.1	23.1	68.3	17.1	17.1	15.8	4.9	21.9	

TABLE 5. *Deficiency Signs Observed among 538 Children and Pregnant or Lactating Women in Institutions.*

GROUP	NO. OF SUBJECTS EXAMINED	NEGATIVE PHYSICAL EXAMINATION	FRONTAL BOSS	HARRISON'S GROOVE	RACHITIC ROSARY	BOW LEGS AND DEFORMITY OF LONG BONES	ENLARGED EPIPHYSIS	RACHITIC POT BELLY	NONSPECIFIC GLOSSITIS	NONSPECIFIC GINGIVITIS	DENUDED LOWER LIP	ANGULAR STOMATITIS	CHEILOSIS	KERATOSIS FOLLICULARIS	SEBORRHEIC DERMATITIS	MODERATE CONJUNCTIVAL THICKENING	CONJUNCTIVAL DRYNESS	MAGENTA TONGUE, WITH FOLLICULAR HYPERTROPHY	BITOT'S SPOTS	CALF TENDERNESS	NERVE TENDERNESS	THYROID HYPERTROPHY	INANITION
Children																							
2 years of age and under....	183	15	119	82	110	12	49	16	43	1	7	11	1	0	6	0	0	0	0	0	0	0	14
3–5 years of age	96	0	80	48	71	24	70	12	51	5	6	7	0	1	4	0	4	0	1	0	0	0	0
10–15 years of age	151	0	70	61	75	9	33	0	113	22	75	31	26	23	18	21	4	0	0	3	6	38	0
Totals	430	15	269	191	256	45	152	28	207	28	88	49	27	24	28	21	4	0	1	3	6	38	14
Percentages		3.5	62.7	44.4	59.3	10.5	35.4	6.5	48.1	6.5	20.5	11.4	6.3	5.6	6.5	4.9	0.9		0.2	0.7	1.4	8.8	3.3
Pregnant or lactating women..	109	2	12	0	0	13	7	0	67	26	46	11	6	32	3	34	0	2	7	9	3	26	0
Percentages		1.8	11.0			11.9	6.4		61.5	23.8	42.2	10.1	5.5	29.4	2.7	31.2		1.8	6.4	8.3	2.7	23.8	

TABLE 6. *Deficiency Signs Observed among 2169 Children and Pregnant or Lactating Women in General Population.*

GROUP	NO. OF SUBJECTS EXAMINED	NEGATIVE PHYSICAL EXAMINATION	FRONTAL BOSS	HARRISON'S GROOVE	RACHITIC ROSARY	BOW LEGS AND DEFORMITY OF LONG BONES	ENLARGED EPIPHYSIS	RACHITIC POT BELLY	NONSPECIFIC GLOSSITIS	NONSPECIFIC GINGIVITIS	DENUDED LOWER LIP	ANGULAR STOMATITIS	CHEILOSIS	KERATOSIS FOLLICULARIS	SEBORRHEIC DERMATOSIS	MODERATE CONJUNCTIVAL THICKENING	CONJUNCTIVAL DRYNESS	MAGENTA TONGUE, WITH FOLLICULAR HYPERTROPHY	BITOT'S SPOTS	CALF TENDERNESS	NERVE TENDERNESS	THYROID HYPERTROPHY
Children																						
2 years of age and under	63	1	47	40	46	5	17	1	17	0	9	6	1	0	6	0	0	0	0	0	0	0
3–5 years of age	278	10	151	130	87	58	107	18	146	6	105	47	33	33	24	9	6	7	0	1	0	22
6–9 years of age	862	42	369	398	243	117	259	0	448	53	432	175	115	116	74	64	3	18	2	5	0	56
10–15 years of age	579	33	184	155	155	54	134	0	311	42	244	103	68	105	77	87	1	19	3	7	0	64
Totals	1782	86	751	723	531	234	517	19	922	101	790	331	207	254	181	160	10	44	5	13	0	142
Percentages		4.8	42.1	40.6	29.8	13.1	29.0	1.1	51.7	5.7	44.3	18.6	11.6	14.3	10.2	9.0	0.6	2.5	0.3	0.7		8.0
Pregnant or lactating women	386	8	71	0	0	22	5	0	317	170	110	48	32	11	31	128	5	2	21	27	9	70
Percentages		2.1	18.4			5.7	1.3		82.1	44.0	28.5	12.4	8.3	2.8	8.0	33.6	1.3	0.5	5.5	7.0	2.3	18.3

TABLE 7. *Estimate of Average Daily Italian Diets, 1930–1944.**

GROUP	MILAN (1930)[17]				MILAN (1936)[18]				CENSUS (1944)[19]			
	TOTAL CALORIES	PROTEINS	FATS	CARBOHYDRATES	TOTAL CALORIES	PROTEINS	FATS	CARBOHYDRATES	TOTAL CALORIES	PROTEINS	FATS	CARBOHYDRATES
		gm.	gm.	gm.		gm.	gm.	gm.		gm.	gm.	gm.
Urban population	2905	95	89	416	3003	93	95	444	2200	78	47	374
Rural population	3153	94	73	513	3272	94	73	560	2700	94	54	441
Averages	3025	95	81	464	3135	94	84	502	2550	86	51	417

*Data based on studies and analyses of weekly food consumption carried out by trained and experienced workers.

The principal Italian dietary alterations associated with war appear to be reduction in dietary fat and carbohydrate, with relative caloric diminution in recent diets. The popularly reported caloric inadequacy of the 1944–1945 Italian ration (900 to 1400 calories) should be interpreted with the realization that approximately only 12 per cent of a person's usual food consumption was derived from ration-card sources, and only about 5 per cent of the weekly food expenditure was utilized for the purchase of rationed food.[19] Seventy-five to 90 per cent of the weekly food expenditure was used for purchase of free or black-market foods. In most areas communal restaurants provided calorically adequate, low-cost meals for families in the lower economic bracket. In Milan, for example, approximately two thousand restaurants provided 600,000 persons with daily meals at a usual rate of 4 to 50 lire per meal. Food was usually available, but there is little doubt that the inflationary economy imposed a severe economic burden on fixed-income groups. The effect of such factors as drought, lack of fertilizer, land mining and political flux on present and future crop production is unpredictable, and the accurate assessment of national food reserves is exceedingly difficult.

The high incidence of clinical rickets — 27.7 per cent — is of some interest. Cod-liver oil and concentrated vitamin D preparations were either in short supply or used for therapeusis rather than prophylaxis. Children were often placed in the vicinity of the sun rather than in direct exposure to its rays — a practice that apparently originated from the experience that direct exposure of infants to the spring or summer sun occasionally induced "convulsions and death." In addition, it is quite possible that dietary calcium intake was inadequate, since the relatively low milk and dairy-product consumption[17, 20] had been further reduced by war. Irrespective of these conditioning factors, the existence and maintenance of numerous hospitals for the treatment of rachitic deformities in children suggest that for generations clinical rickets has been widespread in

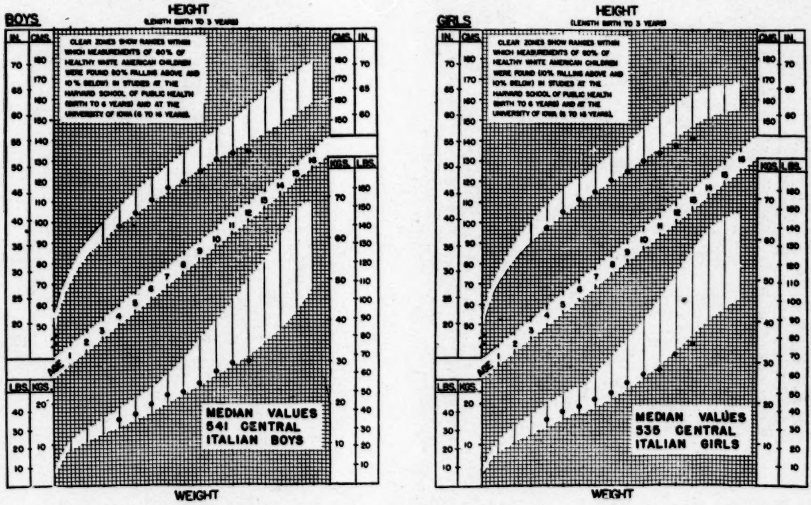

FIGURE 2. *Median Values for Heights and Weights of Central (Rome Area) Italian Children of School and Population-at-Large Groups.*

The values (black dots) are superimposed on charts indicating the 80 percentile ranges for height and weight of normal American children derived from the data of Vickers and Stuart.[24] *Heights were measured in bare or stockinged feet, and weights in light underclothing; ages were taken to the nearest birthday. Data applicable to 231 institutionalized children in the Rome area were not included. Of these, the medians and 80 percentile ranges of 61 males of four, five and six years of age and 36 females of four and five years of age were consistently lower than the medians charted.*

Italy, in spite of its reported prewar incidence of only 0.5 to 1.5 per cent.[21]

Height and weight for age data are often applied as a gauge of grossly inadequate nutrition in children. In France, from 1941 to 1945, Meiklejohn[22] reported evidence of growth retardation in children over seven years of age, whereas in Austria in 1945, Pyke[23] was unable to demonstrate significant changes in the height-weight trend of ten-year-old Viennese children. The data afforded by these surveys suggest no marked deviation in the general population from prewar Italian standards for preschool and school-age children. It is interesting that median values for height and weight of central and northern Italian boys and girls of the weighted samples studied fell, for the major part, within the lower limits of the 80 percentile range[24] for height and weight of normal American school children (Figs. 2 and 3). The values obtained were similar, if not strictly comparable, to those reported for American children of Italian descent and fairly low income groups.[25]

In contrast to those of children in school and in the general population, survey values for height and weight of infants fell consistently below the Italian national prewar levels at approximately the 10 percentile level. Although this probably reflected in part the weighted sampling technic employed, some degree of growth retardation probably existed in the infants two years of age and under (Fig. 4). The relatively greater deviation from prewar standards noted in weight rather than in height is in accord with the observations on infantile dystrophy suggested by Variot.[26] Data on weight at birth were not obtained in these surveys; recent and fairly reliable statistics, embracing southern and central Italian infants, however, suggest that no significant alteration in mean birth weight occurred during the war, although some modal shifts were noted.[19]

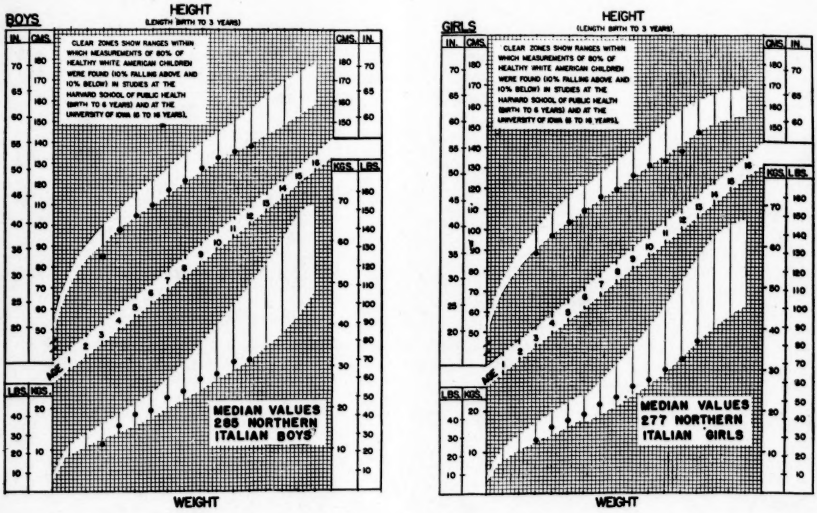
BOYS
HEIGHT
(LENGTH BIRTH TO 3 YEARS)
CLEAR ZONES SHOW RANGES WITHIN WHICH MEASUREMENTS OF 80% OF HEALTHY WHITE AMERICAN CHILDREN WERE FOUND (10% FALLING ABOVE AND 10% BELOW) IN STUDIES AT THE HARVARD SCHOOL OF PUBLIC HEALTH (BIRTH TO 6 YEARS) AND AT THE UNIVERSITY OF IOWA (6 TO 16 YEARS).
MEDIAN VALUES 285 NORTHERN ITALIAN BOYS
WEIGHT
GIRLS
HEIGHT
(LENGTH BIRTH TO 3 YEARS)
CLEAR ZONES SHOW RANGES WITHIN WHICH MEASUREMENTS OF 80% OF HEALTHY WHITE AMERICAN CHILDREN WERE FOUND (10% FALLING ABOVE AND 10% BELOW) IN STUDIES AT THE HARVARD SCHOOL OF PUBLIC HEALTH (BIRTH TO 6 YEARS) AND AT THE UNIVERSITY OF IOWA (6 TO 16 YEARS).
MEDIAN VALUES 277 NORTHERN ITALIAN GIRLS
WEIGHT

FIGURE 3. *Median Values for Heights and Weights of Northern (Florence, Forli, Vechiano and Milan) Italian Children of School and Population-at-Large Groups.*

SUMMARY

A simple, rapid nutrition-survey procedure, dependent on clinical examination of physiologic stress groups, consistent weighted sampling and standardized recording, was used to gather clinical and epidemiologic information on malnutrition in Italy during 1945. Thirty-five surveys, involving widely divergent areas and weighted in favor of demonstrating malnutrition, were performed. A summary of the reported data on 3109 pregnant or nursing mothers and children of refugee, institutionalized,

school, and population-at-large groups suggests that gross malnutrition was infrequent, that rickets was the only nutritional problem of public-health importance, that there was no conclusive evidence — on the basis of observed deficiencies or growth retardation — that wartime food shortage constituted a major health problem, and that the reported data could probably be duplicated in many areas of the United States if the same method of sampling were employed.

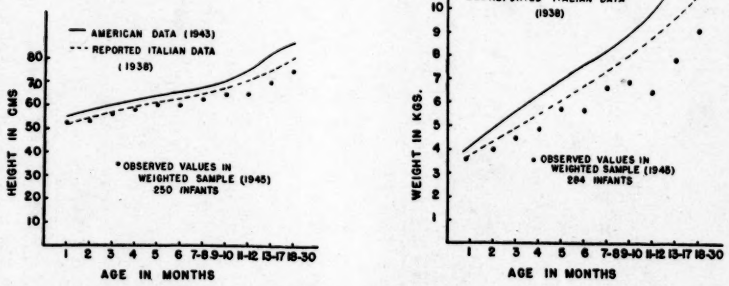

FIGURE 4. *Heights and Weights of Italian Infants Compared with Prewar Italian and American Standards. Infants were examined and measured nude. The height represents recumbent length. Because of the weighted sampling technic employed in all surveys, the height-weight values fall consistently below mean prewar values. The trends of both curves are essentially the same. If samples had been weighted in the opposite direction, height-weight values obtained would be expected to fall at approximately the 80 percentile level of a normal distribution curve.*

We are indebted to Dr. R. E. Butler, Senior Surgeon, U.S.P.H.S., and to Dr. A. P. Meiklejohn for their active interest and field sponsorship of this investigation. The technical assistance of Miss Amalia Baran was invaluable, as was the kind co-operation of many Italian physicians, nurses and government officials. Figures 2 and 3 were devised and furnished by Dr. Harold Stuart, of the Harvard School of Public Health.

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NONTRAUMATIC SPONTANEOUS PNEUMOTHORAX AMONG MILITARY PERSONNEL*

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THIS study was undertaken with the main purpose of determining the etiologic background and the precipitating factors in cases of nontraumatic spontaneous pneumothorax among hospitalized military personnel. Records covering a ten-year period were reviewed. In the six-year interval from January, 1935, to December, 1940, inclusive, when the military strength of the post was relatively low, there were no cases of spontaneous pneumothorax. In the succeeding four years (January, 1941, to December, 1944, inclusive) 41 cases were encountered. The pertinent military, clinical and roentgenologic aspects are presented and discussed.

MILITARY DATA

The incidence of spontaneous pneumothorax for the years 1942 through 1944, based on the annual hospital admissions of military personnel, was practically constant: 0.027 to 0.03 per cent (Table 1). These figures are not valid for comparison with statistics from civilian institutions, since many soldiers were admitted for conditions that would not necessitate hospitalization in civil life.

TABLE 1. *Incidence of Spontaneous Pneumothorax among Military Personnel.*

YEAR	NO. OF HOSPITAL ADMISSIONS	CASES OF SPONTANEOUS PNEUMOTHORAX	
		NO.	PERCENTAGE
1941	48,527	1	0.002
1942	52,947	16	0.03
1943	51,339	14	0.027
1944	32,765	10	0.027

The onset of the pneumothorax ranged from four days to eighteen years after entrance into the Army, and the average length of hospitalization was forty-one days. Twenty-nine soldiers were returned to full duty and 4 to limited duty — a total of 80 per cent. Three patients were transferred to a general hospital, 2 because of failure of the lung to re-expand and 1 (an officer) because of recurrent spontaneous pneumothorax. One enlisted man was separated from the service because of a history of multiple episodes of spontaneous collapse of the lung. Two soldiers were discharged because of an associated psychoneurosis. There were 2 patients with far advanced pulmonary tuberculosis, one of whom died, and the other was transferred to a Veterans Administration hospital.

*From the Medical Service, Regional Hospital, Fort Bragg, North Carolina.

CLINICAL FEATURES

The remainder of this report is based on 39 patients in whom sufficient clinical and roentgenologic data were available for study (Table 2). All were men, and the average age was twenty-four years and ten months. Four soldiers, or 10 per cent, gave a history of an antecedent spontaneous pneumothorax; in only 1 did the recurrence develop in the military service.

Symptoms

It is logical to assume that the intensity of the initial pulmonary symptoms depends on the size of the rupture, the presence or absence of adhesions or fluid, or both, and the type of pneumothorax — whether closed, open or valvular. The onset was sudden in all but 1 case. Shortness of breath, which was a complaint of all patients, was objectively present in comparatively few. Chest pain, of varying severity, occurred in every patient, and the pain was most frequently localized over the affected lung but sometimes radiated to the shoulder, neck, lower back or abdomen. Cough was present in 7 soldiers, cyanosis in 3, mild collapse in 3, and fever in 2 (active pulmonary tuberculosis).

The relation between physical exertion and the production of a spontaneous pneumothorax is not only of medical interest but also of practical importance from a military viewpoint. The following circumstances at the time of onset were studied (in 1 case an adequate history was not available):

At ease or at rest. It is significant that, in 23 cases (59 per cent), the episode could not be associated with any provoking cause.

Physical effort not specifically related to military activity. Ten soldiers, or 26 per cent, were classified in this group. The exertion was mild (walking).

Physical effort directly related to military activity. Five soldiers, or 13 per cent, gave a history of such exertion immediately prior to collapse of the lung. The military duties performed at the time were as follows: hauling sand; kitchen police, with lifting of heavy utensils; shoveling dirt; running over the obstacle course; and drilling troops.

Physical Signs

The classic signs of air in the pleural cavity are well known. Their prominence depends on the extent of pulmonary collapse and of mobility of the mediastinum. The most constant single sign recorded was diminished or absent breath sounds over the pneumothorax pocket. In all cases, the spontaneous pneumothorax was unilateral. The

lungs were almost equally affected: the right in 54 and the left in 46 per cent. None of the patients showed subcutaneous or mediastinal emphysema.

Tuberculin Test

This simple and valuable test was performed in only 12 cases (excluding the 2 patients with active tuberculosis). A positive reaction does not necessarily indicate clinical tuberculosis, but a negative test virtually excludes this diagnosis, provided that there are none of the conditions that would depress the allergic response. Six of the 12 soldiers were positive reactors. Three of these presented no pulmonary lesion on the x-ray film, 2 showed calcified, first-infection-type tuberculosis, and 1, a calcified primary complex with slight linear productive apical infiltration.

Roentgenologic Features

Roentgenography of the chest is the most reliable method of diagnosis of pneumothorax. A film taken at the end of forced expiration is especially valuable in detecting a small collection of air in the pleural space. The findings (Table 2) can be summarized as follows:

Table 2. *Clinical and Roentgenologic Data in 41 Cases of Nontraumatic Pneumothorax.*

Case No.	Age	Length of Service	Hospital Stay	Clinical Data						Roentgenologic Data			
				History of Physical Exertion	Chest Pain	Objective Dyspnea	Other Symptoms	Side Affected	Tuberculin Test	Pulmonary Lesion	Degree of Collapse†	Fluid	Adhesions
1941	*yr.*	*mo.*	*days*										
1*	22	9	8										
1942													
2†	21	26	67										
3	25	1	30	None	Mild	None	None	Right	Not performed	None	+	None	None
4	25	9	54	None	Mild	None	None	Right	Not performed	None (incomplete re-expansion)	+	None	None
5	24	17	36	Severe	Mild	None	None	Left	Not performed	None	+	None	None
6	27	10	29	Severe	Mild	None	None	Left	Not performed	None (incomplete re-expansion)	++	Slight	None
7	25	3	52	None	Marked	Moderate	None	Right	Positive	Calcified primary complex	+++	None	None
8	37	216	29	None	Mild	None	None	Right	Not performed	None	+	None	None
9	23	5	11	None	Mild	None	Cough	Right	Not performed	None	+	Slight	None
10	28	7	30	None	Mild	None	Cough	Left	Not performed	None	+	None	None
11	25	13	35	Mild, not related to military duty	Mild	None	None	Left	Not performed	None	++	Slight	None
12	22	1	31	None	Moderate	Moderate	Cyanosis	Left	Not performed	None	+++	None	None
13	29	7	46	None	Marked	Marked	Cough, fever and physical collapse	Left	Not performed	Bilateral active tuberculosis	++	Small	None
14	29	3	42	Severe	Moderate	Mild	None	Left	Positive	Calcified primary complex and right fibrotic infiltrate	+	None	None
15	24	9	23	None	Mild	None	None	Right	Not performed	Calcified lymph node on right	+	None	None
16	27	26	42	None	Mild	None	None	Left	Not performed	None	+++	Slight	None
17	25	1	87	Mild, not related to military duty	Moderate	Moderate	Cyanosis and collapse	Left	Not performed	Calcified lymph node on right	+++	Slight	None
1943													
18	28	7	40	None	Moderate	Mild	Collapse	Right	Not performed	None	++	Slight	None
19	28	4	20	None	Moderate	Mild	Cyanosis	Right	Not performed	None	+++	None	None
20	20	1	43	Mild, not related to military duty	Mild	None	None	Right	Not performed	None	+	None	None
21	24	18	11	Mild, not related to military duty	Mild	None	Cough	Right	Negative	None	+	Slight	None

Pulmonary Disease

Active, far advanced pulmonary tuberculosis (positive sputum)was found in 2 patients (5 per cent), 1 of whom had a right-sided acute bronchopneumonic process on admission and subsequently developed a spontaneous pneumothorax, with a persistent bronchopleural fistula. The lung showed right side, and ten days before death a spontaneous collapse developed on the left. The left lung, at autopsy, presented miliary and conglomerate tubercles, caseous foci of varying size, multiple cavities and, in addition, several small, thin-walled emphysematous blebs at the apex. It was the opinion of the pathologist that the spontaneous pneumothorax was due to the rupture of an emphysematous bleb (one bleb was found open), rather than to a perforating tuberculous lesion.

Inactive, minimal pulmonary tuberculosis (negative sputum) was present in 3 cases, or 8 per cent. In all, the roentgenogram showed unilateral apical

TABLE 2. *Clinical and Roentgenologic Data in 41 Cases of Nontraumatic Pneumothorax (Continued).*

Case No.	Age	Length of Service	Hospital Stay	Clinical Data: History of Physical Exertion	Chest Pain	Objective Dyspnea	Other Symptoms	Side Affected	Tuberculin Test	Roentgenologic Data: Pulmonary Lesion	Degree of Collapse†	Fluid	Adhesions
1941	yr.	mo.	days										
22	30	1	26	None	Mild	None	None	Left	Not performed	None	+	None	None
23	19	4	17	None	Mild	None	Cough	Right	Not performed	None	+	None	None
24	21	10	95	None	Mild	None	None	Right	Not performed	None	+	None	None
25	33	1	74	Mild, not related to military duty	Mild	Mild	None	Right	Not performed	None	++	None	None
26	25	1	29	Mild, not related to military duty	Mild	Mild	None	Right	Not performed	Fibrotic infiltrate at right apex	+	None	Some
27	19	3	29	Severe	Mild	None	None	Right	Not performed	None	++	None	None
28	28	4	24	None	Mild	None	None	Left	Negative	None	+	None	None
29	22	12	7	None	Mild	None	None	Left	Not performed	None	+	None	None
30	30	22	116	No data available	Mild	Mild	None	Left	Not performed	None	++	None	None
31	22	4	27	Severe	Mild	Mild	None	Left	Not performed	None	++	Slight	None
1944													
32	22	39	41	None	Moderate	Mild	None	Right	Positive	None	+	None	None
33	18	1	98	None	Marked	Marked	Cough and fever	Right	Not performed	Acute tuberculous bronchopneumonia on right	+++	Slight	Some
34	22	54	35	None	Mild	Mild	None	Left	Not performed	None	+++	None	None
35	37	23	66	Mild, not related to military duty	Mild	None	None	Right	Positive	Calcified focus on right	+	Slight	None
36	22	21	11	None	Mild	None	None	Right	Positive	None	+	Slight	None
37	23	27	23	None	Mild	None	None	Left	Negative	None	+	Slight	None
38	26	36	46	Mild, not related to military duty	Mild	None	None	Left	Positive	None	++	Slight	None
39	20	20	35	Mild, not related to military duty	Moderate	Moderate	Cough	Right	Negative	None	++	None	Some
40	19	6	30	Mild, not related to military duty	Mild	None	None	Left	Negative	None	+++	None	Some
41	35	28	52	None	Moderate	Mild	None	Right	Not performed	Calcified lymph node on left and fibrotic infiltrate on right	+	None	Some

*Adequate clinical and roentgenologic data not available.

†The degrees of collapse are as follows: +, 5 to 25 per cent; ++, 30 to 45 per cent; and +++, 50 per cent or more.

only slight re-expansion three months later, when the patient was transferred to a Veterans Administration hospital. The second soldier had bilateral tuberculosis, with cavitation and bronchogenic spread. Artificial pneumothorax was induced on the

linear productive infiltration. The films also revealed a calcified primary complex in 2 cases, in 1 of which the spontaneous collapse was on the side opposite the reinfection infiltrate. These were the pulmonary findings on admission, and nothing new was noted after re-expansion of the involved lung.

Calcified, primary infection-type tuberculosis only was observed in 4 cases (10 per cent), in 3 of which the pneumothorax occurred on the same side. Again, re-expansion showed no other foci.

No pulmonary process could be demonstrated in 30 cases, or 77 per cent. In 28 cases there was no evidence of pulmonary disease or of emphysematous blebs or bullae after total re-expansion; 2 soldiers were transferred to a general hospital after observation for twenty-seven days in 1 and fifty-four days in the other had failed to reveal significant pulmonary re-expansion, although the visualized lungs were clear.

Extent of Pulmonary Collapse

In the initial roentgenogram taken at the end of inspiration the collapse was considered mild (5 to 25 per cent) in 51 per cent, moderate (30 to 45 per cent) in 26 per cent and marked (50 per cent or more) in 23 per cent of the cases; in the last group x-ray films after re-expansion were negative in 6 cases, 2 showed a healed primary lesion, and 1 revealed active tuberculosis.

Pleural-Fluid Formation

No fluid was observed in 65 per cent, a tiny amount covering the costophrenic angle in 25 per cent, and a small effusion up to the tenth posterior rib in 10 per cent of cases. The 2 soldiers with active tuberculosis, as well as 2 with a calcified first infection, were in this last category. The fluid completely resorbed in all but the 2 soldiers with active tuberculosis.

Adhesions

Adhesions were noted in 13 per cent of cases. Two patients presented no pulmonary lesion after re-expansion, 1 had active tuberculosis, and the remaining 2 had inactive minimal tuberculosis.

Treatment

All patients received bed rest, partial or complete. Symptomatic medication to allay apprehensiveness and cough was administered to some. Aspiration of air from the pleural cavity to relieve dyspnea was performed in 3 soldiers, 2 of whom had active pulmonary tuberculosis; these 2 patients were the only ones who also needed oxygen.

Discussion

The problem presented by nontraumatic spontaneous pneumothorax in the Army is not an insignificant one. The important features to be evaluated are etiology, therapy and military disposition.

Etiology

The immediate cause of spontaneous pneumothorax is a tear of the visceral pleura and the subjacent lung. Many pathologic conditions, however, are responsible for nontraumatic pulmonary collapse. To enumerate them would be superfluous. The chief diagnostic problem is the determination of the presence or absence of tuberculosis as the etiologic agent. It is the consensus that, of the various pulmonary afflictions, tuberculosis is the most frequent. On the basis of autopsy reports, about 10 per cent of patients develop this complication in the course of the disease.[1] The highest incidence is in the actively progressing lesions in which a subpleural cavity or caseous focus, which is free of overlying adhesions, erodes into the pleural cavity. The tearing of adhesions at the base of a necrotic lesion also initiates spontaneous pneumothorax. Furthermore, subpleural emphysematous blebs with rupture may develop, especially in conjunction with fibrotic tuberculous lesions. Their coexistence in a given patient renders evaluation of the precipitating factor difficult.

During the last two decades particularly, with the increasing use of chest x-ray examinations, many cases of spontaneous pneumothorax have been detected in apparently healthy persons with no symptoms, signs or roentgenographic evidence of tuberculosis or other pulmonary disease. Most cases have been observed in young men. The causative association, in the great majority of cases, between a so-called "idiopathic pleural effusion" and tuberculosis is still valid. Although the possible existence of a small subpleural tuberculous focus that is not demonstrable by roentgenography may be the source of a pneumothorax, accumulated data indicate that the old conception of the rigid relation between spontaneous pneumothorax and tuberculosis needs revision. Essential, idiopathic and primary spontaneous pneumothorax are terms that are designated as the pneumothorax of apparently healthy persons. Obviously, every case in which such a diagnosis is made should be carefully studied during and after complete re-expansion of the lung to rule out tuberculosis. If the disease has been excluded at the time, the patient is no likelier to develop tuberculosis later than the average person. Kjaergaard[2] observed only 1 case in a follow-up study of 49 patients after a two-year period. Perry,[3] who re-examined 55 out of 85 patients without finding a single case of tuberculosis, stated that in 250 cases mentioned in the literature that were checked for several years only 6 patients had tuberculosis.

In our clinical material, 28 patients, or 72 per cent, definitely showed no clinical or roentgenologic evidence of tuberculosis either of the first or re-

infection type, and they can be grouped as cases of primary spontaneous pneumothorax. In 1943, among the Army population in the continental United States, there were 873 hospital admissions for this condition.[4] The average hospitalization period was forty days. Approximately 15 per cent of the soldiers were separated from the service, and the remainder were returned to general or limited duty. Our figures conform closely to these. The most reliable criteria in differentiating a simple primary from a tuberculous spontaneous pneumothorax are negative clinical and roentgenologic evidence of tuberculosis, a negative tuberculin test, absence of adhesions, little or no fluid, absence of fever and prompt recovery with fairly rapid reexpansion of the lung.

The pathogenesis of primary spontaneous pneumothorax is explained on the basis of one of three main conditions or mechanisms.

Congenital cystic disease of lungs. There may be congenital maldevelopment of the bronchi and cystlike pulmonary anomalies. One or more of the cystlike dilatations may rupture and produce spontaneous pneumothorax.[5]

Rupture of subpleural emphysematous blebs. The most frequent site for these blebs is in the apexes and upper anterior margins of the lungs. The theory has been advanced that under conditions of increased intrathoracic pressure, such as that produced by coughing and sneezing, with the glottis closed, air is forced from the bases of the lungs into the apexes because of the less rigid nature of that portion of the chest wall.[6] The process of repeated distention of the apexes ultimately causes a rupture of the elastic fibers in the walls of peripherally located alveoli. Air then escapes into the alveolar layer of the pleura, which becomes raised and separated from the alveoli. The air dissects itself along the pleura and is finally intercepted by the lobular septums. The mere presence of emphysematous blebs, however, does not necessarily augur the development of spontaneous pneumothorax. Positive autopsy evidence of blebs is often found in patients with a negative clinical history of spontaneous pneumothorax. This accident is attributed to a check-valve mechanism, which permits easy ingress but difficult egress of air from the subpleural bleb, so that the bleb becomes distended and thin walled and finally ruptures, with or without an element of physical exertion. Roentgenographic examination sometimes demonstrates in large blebs or vesicles a fluid level that constitutes indirect evidence of a check valve. Furthermore, at post-mortem examination, the air cannot readily be expressed from some blebs, whereas in others the procedure is comparatively easy. The check valve manifests itself as an expiratory obstructive mechanism in the form of inspissated exudate, scar tissue in the region of the bleb and the adjacent bronchiole (Kjaergaard[7] reported such a finding at autopsy in 2 nontuberculous patients) or openings between the bleb and the communicating narrowed alveoli that may be of different sizes.[8] Emphysematous blebs are sometimes seen in the roentgenograms of the chest, usually in the apexes, and appear as thin-walled ring shadows that simulate tuberculous or bronchiectatic cavities. The absence of surrounding reaction and the lack of clinical symptoms and signs constitute the chief differential features. It should also be stated that thoracoscopy — direct inspection of the pleural cavity in the presence of pneumothorax — may reveal emphysematous vesicles; the best objective ante-mortem evidence is furnished by this procedure.

Rupture of interstitial emphysematous blebs. McGuire and Bean,[9] Hamman[10] and Macklin[11] believe that this is a plausible mechanism. Macklin, in experimental animal studies, found that by increasing the intrapulmonary pressure, air escapes from small ruptures in the walls of alveoli or bronchioles and then traverses the perivascular sheaths through the interstitial tissue to the root of the lung, where it pierces the mediastinum and produces spontaneous mediastinal emphysema, dissects along the fascial planes in an upward or downward direction, or both, — and thus causes a more or less generalized emphysema, — or extends to the pleura forming a bleb that ruptures. Miller[12] recently described 4 cases of spontaneous mediastinal emphysema, 2 of which were associated with a pneumothorax. The simultaneous bilateral spontaneous pneumothorax that occurs in some idiopathic cases is probably due to a rupture in the portion of the posterior mediastinum, between the esophagus and aorta, in which the two layers of mediastinal pleura are in contact. Macklin and Macklin[13] suggest that there may be a constitutional or hereditary factor — an inherent weakness of the alveoli and the pleura — which predisposes to rupture. In support of this hypothesis, they refer to reported cases of spontaneous pneumothorax occurring in the same families, in most of whom there was no demonstrable pulmonary pathology, tuberculous or otherwise, to account for the pneumothorax.

Therapy

Additional remarks on treatment are pertinent.

The immediate treatment of spontaneous tuberculous pneumothorax consists in complete bed rest, relief of shock, if present, symptomatic medication for cough, nervousness and so forth, aspiration of air or fluid, or both, from the pleural cavity to relieve cardiac or respiratory distress and oxygen therapy. A spontaneous pneumothorax should be converted into a therapeutic pneumothorax if the underlying pulmonary disease warrants its mainte-

nance. If, after a reasonable period, the affected lung shows little or no tendency to re-expand, the possibility of a bronchopleural fistula should be investigated. This condition was present in one of our tuberculous patients with a tension pneumothorax, and underwater drainage was instituted, with considerable relief of dyspnea.

Most patients with primary spontaneous pneumothorax require no treatment except partial or complete bed rest for a few weeks. Symptomatic treatment, oxygen therapy and aspiration of air are applicable when indicated. The immediate prognosis in unilateral cases is excellent. Recurrent pneumothorax is not infrequent, however. Its incidence, of course, depends to some extent on the length of the follow-up period of observation. Four of our cases, or 10 per cent, were recurrences. Wilson[14] reported 20 per cent, and Ornstein and Lercher[6] 30 per cent. Recurrences in any given patient cannot be foretold in advance and cannot be prevented except by the obliteration of the pleural space by adhesions. This can be artificially produced, but should not be undertaken after the first episode of spontaneous pneumothorax because the majority of patients suffer only one attack, following which they engage in moderate or strenuous exertion for years with no recurrence. Waring[15] discusses the problem of inducing a chemical pleuritis, by the injection into the pleural cavity of an irritant, such as sterile mineral oil or Gomenol, to create pleural symphysis.

Military Disposition

The disposition, which is not always uniform for any given condition, varies with Army directives, the medical installation in which hospitalization occurs, the particular type of duty performed by the soldier and so forth. The disposition of patients with nontraumatic spontaneous pneumothorax from a medical point of view depends largely on the etiologic diagnosis. Patients with cases secondary to pulmonary tuberculosis (reinfection type) should be discharged from the service.

In primary spontaneous pneumothorax, the underlying pathologic process is benign. Whether the exertion of military duties is an increased hazard remains an open question. A history of strenuous physical effort, as an exciting cause, is lacking in many cases. It would be interesting to obtain data on the comparative incidence of primary spontaneous pneumothorax and its correlation with physical activity among civilians and military personnel of the same age groups. The chief precautionary measure for these patients is the avoidance of exertion, coughing or straining with a closed glottis, which increase intrapulmonary pressure and may precipitate a spontaneous pneumothorax, provided the appropriate anatomic background exists. Holter and Horwitz[16] also cautioned against airplane travel for persons with a diseased pleura. They reported a case in which the spontaneous pneumothorax was interpreted as being due to the change in atmospheric pressure by ascent to 8000 feet. They believed that the fall in pressure from 760 to 560 mm. of mercury increased the tendency for pulmonary collapse, exploited a pleural weakness (emphysematous bleb) and produced a spontaneous pneumothorax. This conclusion was disputed by Engel and Ferris,[17] who in commenting on the case report, stated that the pneumothorax existed prior to or was coincidental to the flight rather than being caused by it, and that the reduction in barometric pressure is transmitted equally and promptly to all body tissues so that the ratio between the intrapulmonary and intrapleural pressures remains the same. Their explanation was as follows:

> Changes in pressure and volume will occur only in gas-containing structures that have inadequate or no communication with the external atmosphere (i.e., middle ear, paranasal sinuses, gastrointestinal tract). As long as there is no air in the pleural space there will be no relative change in intrapulmonary pressure and hence no tendency for the lung to collapse partially. Similarly an emphysematous bleb would not be any more likely to rupture at altitude unless it had little or no communication with intrapulmonary air.

Probably the most frequently recommended military disposition is reclassification of the soldier to limited duty after an episode of unilateral primary spontaneous pneumothorax and separation from the Army for patients with recurrent unilateral or simultaneous bilateral collapse. This policy has not been consistently followed at this installation.

Summary and Conclusions

Forty-one soldiers with nontraumatic spontaneous pneumothorax were admitted to a large army hospital during a four-year period (1941–1944). The pertinent military data concerning the incidence, length of service and hospitalization and disposition are presented.

In 39 cases there was sufficient information for evaluation of the clinical and roentgenologic aspects. The most important single clinical feature was the correlation between physical exertion and pneumothorax; in 60 per cent of the cases the episode was unassociated with exertion, in 26 per cent physical effort was mild and not related to military activity, and in 13 per cent exertion was severe and linked directly to military duties. The main roentgenologic observation was the presence of underlying active pulmonary tuberculosis in 5 per cent, inactive reinfection disease in 7.6 per cent, calcified primary infection in 10 per cent and no parenchymal disease in 77 per cent of the cases.

Primary spontaneous pneumothorax is the pneumothorax of apparently healthy persons with no clinical or roentgenologic evidence of pulmonary disease. Its pathogenesis may be due to rupture of a congenital pulmonary cyst or to a subpleural

or interstitial emphysematous bleb. The differential diagnostic criteria and the therapeutic measures in tuberculous and primary spontaneous pneumothorax are briefly discussed.

The military disposition depends largely on the etiologic diagnosis. Soldiers with pulmonary collapse attributable to tuberculosis (reinfection type) should be discharged from the Army. Primary spontaneous pneumothorax is benign. The disposition most frequently recommended is reclassification to limited military duty following an attack of pulmonary collapse and separation from the service following simultaneous bilateral or recurrent unilateral episodes.

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PLASMA ALPHA AMINO NITROGEN LEVELS IN PATIENTS WITH THERMAL BURNS*

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Increases in the concentration of plasma amino acids following burns have been reported by Glenn, Muus and Drinker[1] in calves, and by Harkins and Long[2] in rats. The present communication reports a study of the plasma alpha amino nitrogen concentration in human beings with extensive burns.

Methods

Twelve patients with severe burns admitted to the Boston City Hospital during the period 1943 to 1945 were studied. The essential details are summarized in Table 1. The area of burn was estimated by the method of Lund and Browder,[3] and the depth of burn classified according to the method of Converse and Robb-Smith.[4] These authors classify the depth of burn by name instead of by degree, using the terms "epidermal" for burns with erythema only, "dermal" and "deep dermal" for superficial and deep injury of the derma without complete destruction, and "deep" for burns with complete destruction of all epithelial elements. In this study the area of epidermal burn was disregarded. The depth of burn cannot be determined with accuracy in patients who survive for only a few days, and consequently can only be approximated. The local treatment was dry, sterile, pressure dressings with no preliminary cleansing. Anesthesia was not given, the dressings being done under mild morphine sedation.

The alpha amino nitrogen concentration was determined by the ninhydrin method of Van Slyke.[5,6] The determinations were usually made on plasma and occasionally on serum, care being taken to avoid delay in separation of the blood cells.

A preliminary study of the method was made on fifty blood samples from 9 normal persons. The mean value for all determinations was 3.6 mg. of alpha amino nitrogen per 100 cc. of plasma, with a standard deviation of 0.36 mg. from the mean. The criterion for significant elevations over normal in the patients under study was a concentration of alpha amino nitrogen at least three times the standard deviation above the normal mean value — that is, a level of 4.7 mg. of alpha amino nitrogen per 100 cc. of plasma was considered significantly elevated.

All the patients received homologous plasma in amounts varying from 0.5 to 6.5 liters during the first forty-eight hours. To rule out the possible effects of plasma administration on alpha amino nitrogen concentration in the plasma, 2 normal subjects were given 1.5 liters of two-year-old liquid plasma in the course of two hours. Serial determina-

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tions of plasma alpha amino nitrogen concentrations before, during and at hourly intervals after the administration of plasma showed no significant changes.[7]

Hemoglobinemia was found in almost all samples during the first twenty-four hours. About 15 per cent of the circulating red blood cells, however, fall in the plasma alpha amino nitrogen concentration below normal. In contrast, 8 of the 12 patients showed a significant elevation of the plasma or serum alpha amino nitrogen concentration in the first twenty-four hours following injury. Seven of these 8 patients had burns, chiefly deep, involving over 35 per cent of the body surface, whereas 1 (Case 1)

TABLE 1. *Clinical and Laboratory Data on Patients with Severe Burns.*

Case No.	Age	Sex	Extent of Burn		Period after Burn	Shock	Plasma Alpha Amino Nitrogen	Quick Prothrombin Test*	Hemoglobin	Plasma Nonprotein Nitrogen	Volume of Urine‡	Result
			Total	Deep								
	yr.		%	%	hr.		mg./100 cc.		%	mg./100 cc.	cc.	
1	42	F	15	10	3½	Present	4.9	21/21	90	28	1100 (first day)	Recovered
					4¾	Present	4.2	—	90	32	1100 (second day)	
					13	Present	4.8	22/22	74	36	2500 (third day)	
					27	Absent	3.5	26.5/25.5	84	31	4215 (fourth day)	
					51	Absent	2.9	—	71	32	2500 (fifth day)	
					120	Absent	2.8	—	56	21		
2	12	F	20	5	1	Absent	3.9	—	93	28	900 (first day)	Recovered
					2	Absent	3.7	—	92	30		
					4	Absent	3.3	—	90	32		
					8	Absent	3.8	—	90	28		
					12	Absent	3.9	—	99	33		
					14	Absent	3.5	—	102	29		
					24	Absent	4.2	—	113	—		
3	75	M	20	6	3	Absent	4.1	18/20	100	20	600 (first day)	Recovered
					8	Absent	3.8	18/20	93	22		
					12	Absent	2.8	—	91	23		
					18	Absent	3.7	18/20	99	26		
					24	Absent	3.5	—	98	30		
4	83	M	35	++	2	Present	4.8	20.5/17	102	36	Low (first day)	Died after 22 hr.
					4	Absent	4.9	—	102	43		
					10	Present	5.9	—	88	50		
					13	Absent	5.6	—	86	64		
5	78	M	35	++	2	Present	7.6†	28/24.5	129	35	105 (16 hr.)	Died after 16 hr.
					6	Absent	5.7	30/24.5	118	36		
					10	Present	6.5	29/24.5	132	—		
					12½	Absent	6.1	33/24.5	120	—		
6	29	M	35	++	1	Absent	4.3	—	141	29	1145 (first day)	Died after 22 hr.
					4	Present	4.8	—	172	29		
					6	Present	6.1	—	169	40		
					13	Absent	5.7	—	152	51		
7	33	M	40	+++	2	Absent	4.0†	21/24.5	108	23	340 (first day)	Died on 10th day
					22½	Absent	3.1	27.5/24.5	115	28	1240 (second day)	
					120	Absent	3.1	—	96	24	2675 (third day)	
											5730 (fourth day)	
											3760 (fifth day)	
8	8	F	50	++	3	Present	4.3	21.5/23	135	34	196 (first day)	Died on 3rd day
					5½	Absent	3.6	20.5/23	110	36	540 (second day)	
					13	Absent	4.4	22/23	112	46		
					19	Absent	3.1	25/24	87	55		
					28	Absent	2.7	27/24	86	41		
9	38	F	50	+++	2	Absent	3.8	—	83	20	200 (first day)	Died after 63 hr.
					7	Absent	4.2	—	112	28	858 (second day)	
					9½	Absent	4.4	—	106	35	150 (third day)	
					14	Absent	4.4	—	112	46		
					20	Absent	5.3	—	111	63		
					25	Absent	4.5	—	91	62		
10	45	M	60	+++	½	Absent	5.2	21/22.5	120	27	70 (16 hr.)	Died after 16 hr.
					4	Absent	5.3	26/22.5	127	32		
					11½	Present	6.1	25.5/22.5	130	44		
11	3	F	70	+++	1½	Present	5.8	—	89	30	10 (6 hr.)	Died after 6 hr.
					3	Absent	5.6	—	101	20		
12	33	M	70	+++	1	Absent	4.4	24.5/25	126	26	458 (first day)	Died after 30 hr.
					7½	Absent	5.7	30.5/25	127	37		
					13½	Absent	5.1	27.0/25	125	46		
					22	Present	6.7	26.0/25	118	58		

*The second figure represents the value of the normal control.

†Determination on serum instead of plasma.

‡The figures in parentheses represent the period during which the sample was collected.

would have had to have been destroyed to account for a rise of the plasma alpha amino nitrogen concentration to the levels considered significantly elevated in this communication. Hemolysis of such a degree did not occur.

RESULTS

The over-all data obtained in the 12 patients are listed in Table 1. In no case was there a significant

had 15 per cent of the body surface burned. The elevation of the plasma alpha amino nitrogen concentration in this last case was transitory and not so great as that in the others.

Clinical shock was present in varying degrees in 7 of the 8 patients. Four of these patients had marked hypotension on entry, and in all the plasma alpha amino nitrogen concentration was elevated at that time. In the other 3 patients hypotension occurred

four, eleven and a half and twenty-two hours, respectively, after entry; in the last 2 patients the plasma alpha amino nitrogen concentration was elevated before the period of hypotension, whereas in the other the elevation occurred for the first time at the time of hypotension. In four patients the level remained high for varying lengths of time after the shock had been successfully treated.

There were 4 patients with no significant elevation of the plasma or serum alpha amino nitrogen concentration, 3 of whom had no shock at any time. The extent of burn was 20 per cent in 2 of these patients and 40 per cent in the other. Another patient, an eight-year-old girl, was severely burned, 50 per cent of the body surface being involved. Shock was severe on entry but thereafter was minimal or absent.

In the 8 patients with significant elevation of the plasma alpha amino nitrogen concentration, 7 showed an elevation of the nonprotein nitrogen of the plasma above 35 mg. per 100 cc. Oliguria was usually present in these cases. In all but 1 patient, however, the rise in the alpha amino nitrogen concentration was observed at times when the nonprotein nitrogen concentration in the blood was still normal. Hemoconcentration was present in almost all the 12 cases but was more marked in the 8 patients with increased alpha amino nitrogen concentrations.

No patient in this series had a history or physical signs of liver or renal disease. One patient (Case 9) was a morphine addict. The icterus index in this patient was 10 on entry and rose to 25 within seven hours after injury, at about which level it remained until death on the third day. Icterus indexes in the other patients in the twenty-four hours after entry were unsatisfactory because of the presence of hemoglobinemia. Later determinations were available only in 1 patient (Case 1) with an elevation of the plasma alpha amino nitrogen, and were normal. Icterus indexes were also normal in the 4 patients who had no significant elevation of the plasma alpha amino nitrogen concentration.

The quick prothrombin time was prolonged in 4 of the 5 patients with elevated plasma or serum alpha amino nitrogen concentration on whom the determinations were made, but were not significantly prolonged in 3 patients with no significant elevation of the plasma alpha amino nitrogen.

Post-mortem examinations were performed on 5 patients who had had significant elevations of plasma alpha amino nitrogen concentration and on 2 who had not. In no case were significant hepatic morphologic changes found.

The following case reports typify the findings in the group of patients showing an increased plasma alpha amino nitrogen concentration.

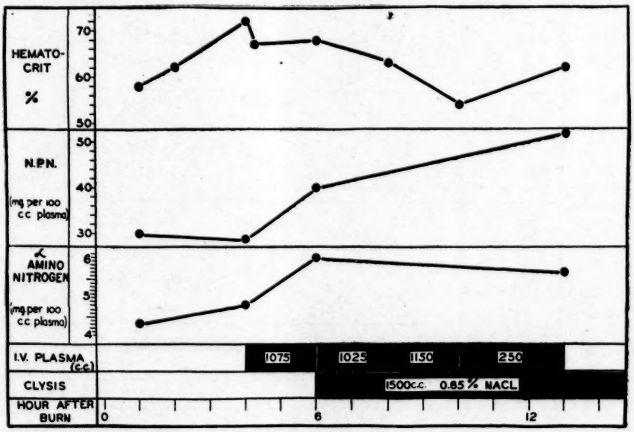

FIGURE 1. *Case 6.*

CASE 6. L. A., a 29-year-old man with a noncontributory history, sustained flame and electric burns and some damage to the respiratory tract shortly before entry. Thirty-five per cent of the body surface was involved in mixed dermal and deep burns, chiefly the latter. The data are presented in Figure 1.

Examination on entry revealed a well developed and well nourished man. The blood pressure was 130/70, the pulse 100 and of good quality, and the respirations 25. The hemo-

globin was 141 per cent, and the alpha amino nitrogen concentration 4.2 mg. per 100 cc. of plasma.

During the next 3 hours small amounts of water were administered orally, no other fluids being given. The patient gradually went into profound circulatory collapse, no blood pressure or pulse being obtainable for the last half hour of this period. The extremities were cold, clammy and cyanotic. There had been no output of urine. The hemoglobin had risen to 172 per cent, whereas the plasma alpha amino nitrogen concentration had risen slightly — to 4.8 mg. per 100 cc. Administration of plasma was then begun, and during the next 2 hours the systolic blood pressure was kept between 80 and 90 and was thereafter above 100. The pulse improved in quality, and the extremities were no longer cold, clammy or cyanotic.

The plasma alpha amino nitrogen had risen to a level of 6.1 mg. per 100 cc. 5 hours after entry and continued elevated during the next 7 hours. The plasma nonprotein nitrogen concentration, which had been 30 mg. on entry, was 40 mg. per 100 cc. at the peak of the alpha amino nitrogen rise and continued to rise to a level of 50 mg. per 100 cc. The patient had occasional periods of respiratory distress, which were relieved by intravenous aminophyllin. The temperature was about 103.5°F.

Twenty-four hours after injury the patient suddenly died from respiratory failure. Autopsy revealed no significant hepatic morphologic changes.

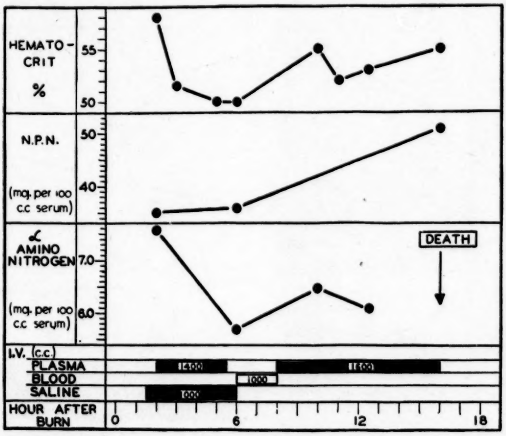

FIGURE 2. *Case 5.*

CASE 5. F. M., a 78-year-old man with a noncontributory history, received flame burns of 35 per cent of the body surface shortly before entry. The burns were mixed dermal and deep. Some damage to the respiratory tract had also been sustained. The data are presented in Figure 2.

Examination on entry revealed an elderly, apathetic man, with cold, clammy, cyanotic extremities. The blood pressure and pulse were not obtainable. The hemoglobin was 129 per cent, the nonprotein nitrogen 35 mg. per 100 cc. of serum, and the serum alpha amino nitrogen concentration 7.6 mg. per 100 cc. Plasma, whole blood and normal saline solution were given intravenously. The hemoglobin varied between 118 and 132 per cent, and the nonprotein nitrogen rose to 50 mg. per 100 cc. of serum. The serum alpha amino nitrogen fell from the initial level of 7.6 to 5.8 mg. per 100 cc. 4 hours later. During this period the blood pressure and pulse were satisfactory. Thereafter, there were 2 brief periods of circulatory collapse, as well as a slight rise in the serum alpha amino nitrogen concentration to 6.2 mg. per 100 cc. There were progressive hoarseness and respiratory distress, and the patient died of respiratory failure 16 hours after injury. Autopsy revealed no significant hepatic morphologic changes.

DISCUSSION

The data show that in most patients with extensive deep burns there was a significant elevation of the serum or plasma alpha amino nitrogen concentration in the first twenty-four hours after injury. This was observed in 7 of 8 patients with burns, chiefly deep, involving 35 per cent or more of the body surface. Marked hemoconcentration and clinical shock were present in all but 1 of these patients. In some patients, however, the rise in plasma alpha amino nitrogen was observed before the onset of hypotension, and in others the elevation persisted after the blood pressure returned to normal. These findings are similar to those reported by Hoar and Haist[8] following tourniquet injury to rats. Elevation of the serum alpha amino nitrogen concentration has also been reported from this laboratory[9] in patients with so-called "medical shock." The correlation of increased alpha amino nitrogen with the severity of shock was marked in these cases.

An increased nonprotein nitrogen concentration in the blood plasma was usually observed in the patients in the present series. In all but 1 case, however, the rise in plasma alpha amino nitrogen concentration occurred at a time when the nonprotein nitrogen concentration was still normal. It has previously been reported from this laboratory[10] and elsewhere[11] that protein catabolism proceeds at a very

high rate in patients with extensive burns. It has generally been accepted[12] that a rise in the over-all nitrogen turnover in the body does not lead to an increase in the plasma alpha amino nitrogen concentration, but when the rate of nitrogen metabolism is markedly increased and the liver is not functioning normally, there is probably some rise in the plasma alpha nitrogen.[13] Although there is no direct evidence that abnormalities of liver function existed in this series of patients, it is likely that some disturbance was actually present. This is suggested by the elevation of the Quick prothrombin time in most cases. Engel et al.[13] and Wilhelmi and his associates[14] have shown in animals that hepatic function is abnormal during hemorrhagic shock, and have specifically demonstrated that the ability of the liver to deaminize amino acids is impaired. Thus, hyperaminoacidemia is probably related to a combination of an increased production of amino acids and a decrease in the rate of deaminization by the liver. This view has also been expressed by Engel et al.,[13] Hoar and Haist[8] and Wilhelmi and his associates[14] to account for the rises in plasma alpha amino nitrogen observed in shocked animals.

Summary

Plasma or serum alpha amino nitrogen concentrations were elevated in 8 of 12 patients with thermal burns.

There was a close correlation among the elevation of the plasma alpha amino nitrogen concentration, the extent of the burn, the degree of shock and the prolongation of the Quick prothrombin time.

In some cases the elevation of the alpha amino nitrogen occurred before marked hypotension was present, and persisted after the blood pressure returned to normal.

The rise in plasma alpha amino nitrogen concentration occurred before that in the nonprotein nitrogen concentration.

It is postulated that hyperaminoacidemia is the result of an increased production of amino acids, together with a decrease in the rate of deaminization by the liver.

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MEDICAL PROGRESS

OCCUPATIONAL DERMATOSES (Concluded)

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BOSTON

VITAL AGENCIES

Bacterial Infections

Lebowich, McKillip and Conboy[37] report 3 cases of cutaneous anthrax. All the patients were working in leather tanneries, where they handled imported skins. After an average incubation period of one to three days they developed anthrax of the skin, which began as a single, small, red papule on an exposed surface that enlarged rapidly, with development of a central necrotic brownish-black eschar surrounded by a pink, doughy swelling and pink vesicles. No pain was experienced but a sense of fullness and numbness at the site of infection. The constitutional symptoms were persistent headache, fatigue and weakness and also fever.

Ecthyma contagiosum, which is an infection frequent in animals, is reported by Kingery and Dahl[38] as occurring in a farm worker who treated sheep for so-called "fluke disease." The animals' mouths were forcibly opened, and in this process the fingers were traumatized. Many of the animals had scabs or pustular lesions on the lips. Three lesions developed on the right thumb, and there were similar ones on the fingers of each hand. The lesions, which were firm papules or papulovesicles on an erythematous base, cleared within a week with mild treatment.

Mycotic Infections

One of us (J. G. D.[39]) has shown that persistent eruptions on the hands, frequently labeled fungous infections by industrial physicians and inexperienced dermatologists, are usually due to secondary infection by pyogenic organisms. Ayers and Anderson[40] state that without confirmatory evidence a diagnosis of fungous infection of the hands stands an 88 per cent chance of being wrong. A positive diagnosis can be made only through laboratory evidence.

The hands and feet of 406 patients with a contact dermatitis of more than three months' duration were examined by a trained mycologist working under the supervision of one of us (J. G. D.[39]), because it is often said that fungus is a secondary invader and prolongs contact dermatitis. Direct microscopic examination of scrapings showed fungous infection on the hands of 5 patients, and cultures showed infection on the hands of 2.

Superficial and deep-seated mycotic infections affect workers. When fungi are regularly found in an occupational contact, it is reasonable to assume that the worker suffering from organisms peculiar to his labor has an occupational disease. Among workers exposed are those who till the soil or come in contact with plants, hay, straw and animals and their by-products, such as feathers, silk, wool and leather. Kennel keepers acquire microsporosis. Dairymen and herders suffer from trichophytosis due to an ectothrix. Dermal moniliasis occurs in bakers, preserve packers, fruit handlers, salad makers and dishwashers. Deep-seated infections are usually found in farmers, cattlemen, florists, horticulturists, wood handlers and research workers. In Massachusetts primary fungous occupational infections are comparatively rare, but industrial physicians should not only know the fungi to which the worker is exposed but also be trained to prepare and to recognize cultures of fungi.

The most striking report of recent years is that of du Toit.[41] In South Africa in 1927 sporotrichosis made its appearance in the gold mines of the Witwatersrand when 14 native workers were found to be suffering from infections due to *Sporotrichum beurmanni*. In 1941, 74 cases were reported in two other mines. All these patients were timber boys. From the middle of 1941 to February, 1942, a total of 650 cases of sporotrichosis was found in an underground shaft where 2500 natives were employed. The epidemic then began to spread among European workers. The company accepted responsibility on the assumption that the disease always followed injury, no matter how insignificant it might be. The clinical types occurring in the mines included the classic lymphangitic type, seen in America, and the multiple ulcerative type, seen in France. The infection was undoubtedly brought into the mine by a native from the tropical east coast. The fungus was isolated from the air and timber in the mines. Treatment, which was simple and effective, consisted of large doses of potassium iodide by mouth, to a total of 200 gm.

Peck and his associates believe that dermatophytosis and its allergic manifestations are not an important factor in lost time among industrial workers.[42] In the report of the Council on the Treatment and Prophylaxis of Dermatophytosis,[43] based on the work of the United States Public Health Service, it is stated that it appears reasonable to assume that more than 50 per cent of persons in industry are affected by dermatophytosis, but it is also affirmed that the evidence of Peck and his associates is most

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important in the dispute over the question whether the allergy of dermatophytosis predisposes to the development of contact dermatitis. Both groups concluded that it did not. These two statements are contrary to our experience in private industrial practice and to the opinion expressed by Neal and Emmons.[44] Examinations in offices and at plants showed that less than 5 per cent of workers demonstrate clinical evidence of fungous infections. We also believe that the role of cutaneous sensitization is well established. The mere presence of dermatophytids on the hands produces sites of lowered resistance sufficient to break the skin and allow the mechanical and chemical contacts of industry to aggravate the original eruption.

Conant et al.[45] describe a long list of mycotic infections, of which the following may be classed as attributable to occupational contacts. Actinomycosis is found in agricultural workers and is twice as frequent in men as in women. North American blastomycosis is presumably not contagious but has been acquired by direct contact, the cases reported being those of a physician infected while performing an autopsy and an infant infected from a cutaneous lesion on the arm of a nurse. The sex ratio is 9 males to 1 female, and the disease is found mostly among the poorer classes. Coccidiodomycosis is an infection carried in the soil and by wild rodents; laborers are likelier to develop the severe form than workers in other occupations, and laboratory workers become infected by inhaling spores from dried cultures. A recent study of the primary form of this disease in Army camps showed that 0.2 per cent of the cases developed into a progressive and fatal form. South American blastomycosis also occurs in a ratio of 9 males to 1 female; the disease develops most frequently in manual laborers whose work is of such a nature that vegetative material is brought into contact with the skin. Chromoblastomycosis affects the laboring classes almost exclusively. Moniliasis causes lesions of the hands in housewives, bakers, waiters, bartenders and fruit packers whose hands are macerated by frequent soaking in water. Sporotrichosis is apparently acquired from plants, although it may be contracted from infected animals. The disease occurs oftener in men than in women, especially in farmers, laborers and horticulturists. Maduromycosis is also more frequent in men than in women; in 100 cases cited by Bocarro,[46] 91 of 92 men were farmers and 7 of 8 women were wives of agriculturists. Aspergillosis is an infection occurring in persons often exposed to massive doses of spores, as in squab feeders who take grain onto their mouths to moisten it, fur cleaners who use rye flour as a grease remover and agricultural workers exposed to dust from threshing machines. Certain species of mucoromycosis produce epidemics of paronychia among orange workers. Rhinosporidosis has been reported only 13 times in the United States; the disease develops in persons who swim or dive in stagnant water and is frequent among the sand divers of India.

Insect Infestations

Saunders[47] reports 2 cases of workers handling straw who developed an itchy eruption. Examination of the straw revealed tyroglyphid mites. The cutaneous manifestations were excoriated urticarial papules, splotchy erythema and intense itching of the neck, face and forearms.

Dermatitis from fowl mites occurring in 2 persons in the same household is reported by Knotts.[48] *Dermanyssus gallinae* was identified as the cause. The patients' clothing was infested with these mites while they were gathering eggs, tending chickens and cleaning the coops. The bites on the skin became papules with pruritus, which on scratching became excoriated and then crusted. Such patients complain of nocturnal itching, because these mites bite at night. This condition is confused with scabies, but the presence of mites on the clothing, the lack of burrows and the patient's occupation serve to differentiate the two conditions.

In a review of occupational dermatitis,[32] so-called "grocer's itch" occurring in handlers of dried fruits is mentioned. Dried dates, figs, prunes, apples and pears are sometimes infested with mites known as *Carpoglyphus passularum* and *Glyciphagus domesticus*. Tea leaves are sometimes infested with a mite known as *Rhizoglyphus parasiticus*, and wheat may contain a mite known as *Aleurobius farinae*.

Chemical Agents

The group of chemical agents causing dermatoses is a large one, comprising the acids, alkalies, salts, hydrocarbons, crude coal-tar products and other organic compounds.

Acids

Schwartz[49] reports an unusual form of occupational dermatitis that occurred in a plant manufacturing hydrochloric acid. The outbreak occurred after a change was made in the manufacturing process, consisting in the addition of 0.4 per cent of soft coal to the salt briquets from which the acid was made. One of the towers in which hydrochloric acid gas was absorbed by water was cleaned out, and all of 13 men thus engaged developed dermatitis after coming into contact with a yellow, waxy deposit on the fan. Eight months later 27 men who cleaned out another tower also developed dermatitis. The eruption began with itching and burning, and edema of the face and eyelids. After the eruption had subsided, there remained comedones, pustules and acnelike cysts. Patch tests with the waxy substance, which contained 5 per cent hydrochloric acid by weight, were positive. This is a primary irritant.

Alkalies

An eczematoid eruption affecting the back of the hands and the flexor surface of the forearms is reported by Whitwell[50] as arising from the carbonizing of steel. During this process organic carbonizers are mixed with sodium bicarbonate or barium carbonate and the impregnated pellets are packed by hand around the steel. The cause of the dermatitis is the sodium bicarbonate in the pellets, which even after being washed are still strongly alkaline. The barium carbonate is not so harmful. The rash developed after workers had been thus employed for several months. Such workers should do no carbon packing when they are sweating or when their arms are wet after washing; otherwise they soon have a strong solution of soda on their skin.

Dural

Hall[7] reports 10 cases of dermatitis due to dural, representing 5 per cent of 202 cases of occupational dermatitis in aircraft workers. Dural is an alloy composed of 95 per cent aluminum, 4 per cent copper, 8.5 per cent each of manganese and magnesium and traces of iron and silicon. The eruption consists of scattered pale-pink, fine papules, usually excoriated and covered with sanguinous crusts. The lesions are located on the forearms. In 1 of these cases they were on the flexor surface of the wrists; the sides of the neck were affected in half the cases. The hand and fingers were not involved. Each of the patients showed a slight or moderate reaction to patch tests with dural drillings. The duration of exposure before evidence of sensitization appeared averaged slightly over five months.

Aluminum

In this same series Hall[7] encountered 4 cases of dermatitis due to aluminum. Each patient showed a slight reaction to aluminum filings. This eruption is evanescent and may disappear within twenty-four hours away from work. The lesions are wheal-like and erythematous, appearing on the forearms, wrists, face and neck. The average duration of exposure is four and a half months.

Selenium Dioxide

Selenium dioxide is used extensively in the electric industry. Workers with pure selenium did not develop skin trouble, but those working with the dioxide compound had typical reactions.[51] A crystal of selenium dioxide under the fingernail produces a painful reaction lasting for about two days. The pain then subsides somewhat, and a small red spot remains. This is followed by a small scar in the area. In severe cases necrosis develops in areas of contact with this compound. There is a burning sensation, intolerable at times. In the series reported, patch tests with pure selenium produced no reaction, but those with selenium dioxide caused such severe reactions that the patches were removed after a few hours. The reactions were painful, and vesicles developed at the site tested, with eventual deep necrosis. This report proves the danger of patch tests with chemicals that are undoubtedly primary irritants.

Cobalt

Cemented carbides are made from tungsten, tantalum and titanium carbides, carbon and metallic cobalt. In a plant manufacturing these materials, 20 men who had worked in the metal room for a month or more before developing dermatitis were examined.[52] The eruption was erythematous and papular. Some lesions were ringed, with clear centers, appearing on the cubital spaces, the sides of the neck, the eyelids, the flexor aspects of the forearms and the backs of the hands. In 2 workers who swept the dust from the floor, patches of dermatitis appeared on the external malleolus and ankle. Patch tests on 6 workers showed negative reactions to oxides of tungsten, tantalum and titanium and to carbon but positive reactions to metallic cobalt and to the black powder containing all the metals. Two controls, not working in the plant, gave negative reactions to all metals and thus showed that this is a sensitivity dermatitis. Some of the workers developed tolerance and were able to continue in this work.

Silver

In a case of argyria reported by Wigley and Deville[53] the black pigmentation of the skin was due to constant exposure to silver nitrate.

Beryllium

In the last four years, 170 cases of beryllium poisoning have been seen in three plants.[54] The manifestations included dermatitis, chronic skin ulcers and inflammatory changes in the respiratory tract. The severest manifestation was a diffuse pneumonitis, which caused the death of 5 workers. In the processing of ore, beryllium oxide is extracted under a high temperature from crushed inert ore beryl by acids and fusion methods. Fumes and dust encountered at various steps in this process were the cause of the poisoning. Contact dermatitis and skin ulcers occurred in 42 workers, usually accompanied by rhinitis and nasopharyngitis. The dermatitis occurred within a week after starting work involving contact with beryllium sulfate, beryllium fluoride or beryllium oxyfluoride. The eruption, which was edematous and papulovesicular, occurred on the hands, arms, face and neck, with intense itching. Ulcers were caused by a beryllium crystal deposited in the skin: a small indurated papule resulted and progressed to necrosis. Early excision and curettage of the site afforded complete healing.

Sodamide

A case of acute sodamide poisoning with a fatal result is reported by Pozhariskii and Khodzhash.[55]

A drum containing the compound burst, igniting the clothing of a girl employed in unloading drums. The flames were soon extinguished, but the portion of the skin in contact with the sodamide suffered severe burns. The patient died the next day. Autopsy showed acute fibrinotic inflammation of the larynx, trachea and bronchi and many local second-degree burns on the skin.

Methyl Bromide

Butler, Perry and Williams[56] report a case in which 2 men whose boots were in contact for five hours with methyl bromide, a fire-extinguisher fluid, developed large blisters of the feet. Wyers[57] calls attention to the symptoms of toxicity of this compound. Brief exposure to the gas caused headaches, smarting of the eyes, cough, loss of appetite, abdominal discomfort and numbness of the feet. Methyl bromide is used in industry as a methylating agent, for refrigeration purposes, as a fumigant and for the control of rodents and fleas. The liquid form rapidly penetrates the clothing, even boots and rubber covering. The characteristic skin lesion is a large blister.

Zinc Chromate

Hall[7] reports that of 132 persons sensitive to zinc chromate primer, 90 (68 per cent) were sensitive to zinc chromate only. The average duration of exposure before the eruption developed was seven months. Nummular eczematous plaques or groups of papules appeared on the radial and flexor aspects of the wrists, the dorsal aspects of the proximal portions of the thumbs and the volnar and ulnar aspects of the forearms.

Lead Arsenate

Monahan[58] reports an unusual case in which a laborer handling bales of dried pumice in a vinegar factory developed contact dermatitis from the dust, which showed a strongly positive qualitative test for arsenic. It was found that the apples whose peelings were used in the manufacture of vinegar had been sprayed with lead arsenate before being dried.

Vinyl Carbazole

Exposure to vinyl carbazole, according to Tabershaw and Skinner,[59] resulted in dermatitis in two plants, where the compound was used in manufacturing electric equipment. It is employed in the polymerized form after 98 per cent pure carbazole is treated with acetylene under heat and pressure and filtered, purified and dissolved in monochlorobenzene in the presence of a catalyst. The white amorphous substance is insoluble in water but soluble in chlorinated hydrocarbons, petroleum hydrocarbons and fats.

Tricresyl Phosphate

Gaul and Henneger[60] report a case of dermatitis bullosum produced in the seborrheic areas of the body by contact with tricresyl phosphate, which was used as a substitute for plasticizer for adhesive tape. The dermatitis was not found in the area of contact, but disappeared on removal of the tricresyl phosphate and recurred when further patch testing was done. The authors stress the significance of a chemical that can invade unbroken skin and produce cutaneous lesions indistinguishable from established disease patterns of seborrhea.

Trichlorethylene Gas

A case of widespread dermatitis due to the inhalation of trichlorethylene gas is reported by Baker and White.[61] There have been no previous reports in the literature of such dermatitis from the inhalation of vapor. A foreman whose office was twenty feet from a degreasing unit suffered diffuse erythematous eruptions of the entire body. The lesions disappeared when he was away from his work but reappeared on his return. When he wore a gas mask no dermatitis occurred, but when he failed to wear it severe burning and pruritic eruptions developed within a few hours. One of us (J.G.D.) had a patient who on exposure to trichlorethylene developed a herpetic eruption of the face and loss of sensation of taste.

Benzidine

Baer[62] reports a recurrent pruritic eruption on the hands and face of a physician who performed benzidine tests on stool specimens. It recurred each day that he performed the test, the eruption starting on the fingers of the left hand. Patch testing showed benzidine to be the cause, benzidine powder producing an extremely strong eczematous reaction.

Explosives

The explosives causing most cases of dermatitis are tetryl, TNT (trinitrotoluol), fulminate of mercury, ammonium picrate and picric acid, smokeless powder, amatol, amonal, hexite, lead styphnate, sensol and lead azide. Nitroglycerin does not produce dermatitis but can be absorbed through the skin and cause cardiovascular disease.

Tetryl. About 4 per cent of tetryl workers develop dermatitis,[5] but most cases occur in the loading areas. Tetryl manufacture and pellet production are to some extent closed processes, which accounts for the small number of cases of dermatitis. Peck, Gant and Schwartz[6] report higher percentages of tetryl workers developing dermatitis. Allergic dermatitis is caused oftener by tetryl than by any other explosive.[19, 63, 64] The eruption appears in newly employed workers within one or two weeks on exposed surfaces — on the face, hands and forearms — and secondarily where dust collects — on the wrists, back of the neck, ankles and feet. The face, hair and hands are stained yellow. Associated symptoms are epistaxis, dryness of the throat and cough.

TNT. Sensitization dermatitis is produced by TNT but not so frequently as by tetryl. The derma-

titis chiefly affects the hands. The cutaneous manifestations are edema, papules and deep-seated vesicles, especially on the palms.[19] Other regions frequently affected include the wrists, forearms, neck creases and feet.[64] The face is rarely involved. There is considerable staining of the hands.

Fulminate of mercury. This explosive, which is more sensitive than tetryl and is used in detonators and primers, causes ulcers of the skin if it enters abrasions and also sensitization dermatitis.[19] The eruption appears first on the backs of the hands and the forearms as erythematous lesions, which tend to remain discrete, and on the face as erythema, with edema of the eyelids and also conjunctivitis.[64] The irritation develops on the fourth to the sixth day of contact.

Ammonium picrate and picric acid. These explosives are used as bursting charges. They cause yellow discoloration of the skin and hair and a sensitization dermatitis of the face.[19]

Smokeless powder. This powder contains dinitrotoluol or diphenylamine, which produces a sensitization dermatitis.

Synthetic Rubber

When the sources of natural rubber were cut off, there was a tremendous increase in the manufacture of synthetic rubber. This process involves several compounds that cause irritation and sensitization dermatitis among workers. Types of synthetic rubber made in this country are buna S, buna N, butyl rubber and neoprene. Comparatively little dermatitis occurs in the manufacture of synthetic rubber, despite the many irritant chemicals employed.[65] This is explained by the facts that factories are now equipped with mechanical safety devices and that most of the chemical processes are closed. The various compounds used and their effects are as follows.

Butadiene. The vapors of this compound are irritating to the eyes, nasal passages, throat and lungs.[66]

Tertiary butyl catechol. This compound is an inhibitor, preventing butadiene and styrene from polymerizing while in storage. It is a skin irritant and causes sensitization dermatitis.[19, 65]

Styrene. This is also a skin irritant and sensitizer.[65]

Phenylbetanaphthylamine. This antioxidant is a skin sensitizer.[65]

Hydroquinone. This compound is usually added to the latex. It is a cause of sensitization dermatitis.[65]

Aluminum chloride. This chemical, which is a catalyst, is a skin irritant.[65]

Bardol. This is a coal-tar derivative and is both a primary irritant and a photosensitizer.[19, 65]

Chlorobutadiene. This liquid causes erythema of the skin from splashes, even if immediately washed off with water.[65]

Acrylonitrile. This compound is extremely toxic, both from vapor inhalation and from toxic absorption. It may cause dermatitis.[65]

Soapstone. This substance is used to cover bales of rubber. If it is coarsely powdered its handlers develop dermatitis, especially during the hot months.[65]

Rubber Goods

Mercaptobenzothiazole. This accelerator was found to be the most frequent cause of sensitization dermatitis.[67]

Rubber gloves. Three cases are reported of physicians who wore rubber gloves and developed itch and dermatitis.[68] Neoprene gloves were substituted for ordinary latex and were tolerated.

Military Cases

A temporary sensitivity that was specific for woolen clothing has been reported in members of the Canadian Army who had previously been treated several times for scabies with lotions whose principal ingredient was benzyl benzoate.[69] Two separate lotions were used, but this compound was the only substance common to both and each lotion produced the same result. Patch testing with wool and cotton showed sensitivity to wool only. In no case was the period between use of the lotion and development of wool sensitivity less than two weeks. In some cases discharge from the military service was necessary. This dermatitis should be distinguished from recurrence of scabies.

Dermatitis from contact with a Navy identification tag has been reported.[70] After forty-eight hours contact with one of these tags, which are made of chrome steel, a steward's mate developed pruritus, burning, marked erythema and vesicles. There was no history of allergy other than urticaria following the ingestion of grapefruit or lemon. The original contact was with the wrist, but the ankle and neck reacted similarly when brought into contact with the tag. The lesions, which developed after two weeks of contact, lasted for over eighteen months. There were no generalized symptoms of chrome poisoning. Excessive sweating may have resulted in greater solubility of chromates as a factor in this sensitivity. No case of this kind had previously been reported.

Dermatitis from the wearing of Army spectacles has been reported.[71] Six cases were seen within three months at a clinic in Scotland. The frames were made of an alloy of nickel, zinc and copper. Patch tests with pure nickel and with alloy plated with nickel gave positive reactions. Tests showed that under conditions of wear in the presence of perspiration and body heat the dissimilar metals of the alloy can set up an electrolytic action and give rise to an allergic phenomenon. In all cases removal of the spectacles brought rapid clearing of the lesions. All the patients but 2 had pityriasis sicca capitis and perspired freely. Prausnitz–Küstner tests were negative, probably because the subjects did not sweat enough. Covering of the spectacle frames

with rubber or some other material prevented dermatitis.

Miscellaneous Cases

Sterling[72] reports a severe dermatitis of four years' duration in a waitress. The areas affected were the flexor surfaces of the forearms and the fingertips; the fingers were either split or showed oozing crusted patches. A germicidal powder known as Microlene was found to be responsible, the patient having handled dishes washed in this chemical solution.

Nine cases of contact dermatitis in a morphine factory were reported by Dore, Thomas and Green.[73] The average period between the beginning of employment and the appearance of dermatitis was fourteen and a half weeks. The eruption began as an irritating erythematous dermatitis of the eyelids, sometimes with edema. The nape of the neck and the area below the chin were next involved in women, and the collar area in men. The specific sensitizer was not found.

According to Goodman,[74] cold-wave "permanents" affect the molecular structure of the skin as well as the hair. This is a simple chemical reaction, not an allergic one. Thioglycolic acid should be suspected when one encounters a dermatitis consisting of erythema, vesicles, fissures, pustules, lichenification and itching of the hands of workers dealing with cold-wave fluids. Commenting on an article by Cotter,[75] however, McCord[76] makes the following observations: there is no primary skin irritation from thioglycolic acid or alkaline derivatives at levels below 8 per cent and no evidence of sensitization and allergic responses with properly prepared materials under any circumstances; hydrogen sulfide formation can be controlled through stabilization; and no measurable systemic responses occurred in his tests on animals after parenteral introduction.

A baker complaining of a skin eruption of one year's duration was found to be sensitive to benzoic acid in the improved flour he was using.[77] When he substituted nonimproved flour, the dermatitis, which affected the hands, neck and chest, rapidly disappeared. A patch test with 6 per cent benzoic acid in liquid petrolatum gave a positive reaction.

A month after administering penicillin to hospital patients a physician developed a dermatitis on the skin of the upper extremities and face.[78] A chemist was observed to have contact dermatitis a year after beginning work with sodium penicillin.[79] Patch tests with this compound had to be discontinued after twenty-seven hours because of itching and burning. One of the 2 cases of allergic reaction to penicillin reported by Barker[80] occurred in a medical officer who handled the drug under laboratory conditions for several months. He spilled it on his hand, and itching of the face developed on the following day. Later an acute dermatitis of the face and neck occurred. A patch test with penicillin gave a positive reaction in twelve hours.

Diagnosis

The diagnosis of occupational dermatosis is based on the history, the sites of the eruption and its characteristics. Careful history taking cannot be overstressed. The history should contain information concerning the length of time the worker has been engaged in a particular job, the time and place of the eruption and the characteristics of the original lesion. A history of a similar eruption in a fellow worker may be helpful but is often misleading. A knowledge of the industrial processes involved is invaluable to the physician, but the rapid increase in new processes makes it impossible for any one person to possess such knowledge. The physician should therefore request advice from the employer, the insurer, the safety engineer and the chemist.

Many report forms have been tried by one of us (J.G.D.), but they are for the most part of little value. Peck[81] proposes a new form for reporting industrial dermatitis. It is fairly comprehensive and might be of value in a single plant, but for general use it takes too much for granted.

Occupational dermatoses usually begin on the exposed parts of the body — the hands, fingers and forearms if the offending substance is a solid or a liquid, and the face or neck if a vapor is involved. In dusty occupations, a dermatitis often starts in the flexural areas above the tops of the shoes. Having the patient strip before examination prevents many an embarrassing situation. Since the skin can express itself in only a limited way, dermatitis tends to manifest itself in the lesions previously described, whether or not it is occupational, but some of the eruptions are characteristic of the irritant involved. For example, acnelike lesions of the face are a characteristic eruption of the chlorinated napthalenes[16]; folliculitis, comedones and furunculosis of the hairy parts of the body are seen in cutting oil eruptions[16]; paronychias occur in fruit handlers[32]; a characteristic staining of the skin is caused by explosives; and the helmet type of dermatitis is produced by fumes, vapors and dusts.

Differential Diagnosis

A sound knowledge of dermatology is a prerequisite for correct diagnosis. Occupational dermatoses must often be distinguished from such diseases as seborrheic dermatitis, fungous infections, lichen planus, impetigo contagiosa, pityriasis rosea, neurodermatitis and contact dermatitis arising from nonoccupational activities. Dermatitis from shoes[82] or wearing apparel[82] must also be differentiated. Some diseases that are normally not occupational — such as fungous and pyogenic infections, keloids from burns, radiodermatitis, keratoses and even cancer — may arise from occupational exposures. The prime requisite in the diagnosis of occupational dermatoses is a knowledge of the principles of dermatology, so that one may classify the eruption presented. Next in importance is an accumulation of

facts regarding the industrial exposure, and last comes a good measure of common sense. If a worker's hands are wet during the greater part of his working hours, if he is constantly exposed to irritation from fumes and dusts, if an epidemic of eruptions appears in a group of workers, if the skin is splashed with a primary irritant, and if the clothes are soiled for hours with a volatile solvent such as gasoline, it requires no great learning to assume that the worker is suffering from an occupational dermatitis. Careful history taking limits the possible factors, and proper application of a few patch tests solves the problem when the history of the irritating factor is not too evident.

The Patch Test

Patch testing consists in putting a portion of the material to be tested on the subject's skin and covering it first with linen and next with a larger piece of adhesive or other impervious material. Although the actual technic is simple, knowledge of when to perform the test, with what materials and in what dilution presents a problem for the expert. Serious consequences can follow the test. Primary irritants should not be used, since some of them are also sensitizers. The latter, in the proper dilution, can be tested. Textbooks usually give tables for guidance. As a rule, products made for wear can be tested full strength or as they are.

It must be realized that because patch testing cannot simulate all the actual conditions of exposure — sweating, friction and other factors to which a worker is subjected — its value is limited. It is an aid in the diagnosis of occupational dermatosis. A recent questionnaire on patch tests[84] supports the view that although these tests have their limitations, they are of definite value. During a twelve-month period, for example, Howell[24] was able to prove the exact cause of 250 cases of contact dermatitis by patch-testing with the suspected materials.

Patch-test reactions are recorded as follows: a + reaction shows definite erythema; a ++ reaction shows erythema and papules; a +++ reaction consists of vesiculation; and a ++++ reaction shows a denuded area at the site of contact. A preplacement patch test on the skin is carried out with materials or chemicals that a worker comes in contact with on his job. The advantages of this test enumerated by Keil[85] consist in eliminating cases of latent hypersensitiveness, placing a person in the right industry and determining whether a substitute chemical should be used. The following objections have been raised to this type of testing: a person may be sensitized to the material tested; he may sue; he may have a positive reaction and yet show no clinical evidence of dermatitis; and evidence of sensitization is not always a sure indication that the worker will suffer a dermatitis. It has been proved that many workers, such as bakers who have never had a cutaneous eruption, react to patch tests with substances to which they are exposed. Again, many workers handle sensitizers for twenty years or more before suffering from a cutaneous disturbance. In still other cases those who continue their work and subsequently become immune or less susceptible are shown by patch tests to be still sensitized, but are able to maintain tolerance to their exposures.

So-called "prophetic patch testing" is done with the purpose of foretelling whether a substance will or will not produce a dermatitis.[83] The test is made on 200 or more persons in the usual way. Since the chemicals to be tested are new ones, it is assumed that there has been no contact with them. Two series of patch tests are carried on in the same subjects ten to fourteen days apart. The first series give reactions to primary irritants or produce them in persons previously sensitized, and the second show how many subjects have been sensitized by the first series.

Legal Aspects

In Massachusetts an occupational dermatosis may be defined in the words used by one of the courts in describing personal injury as "any injury, damage, harm or disease of the skin which arises out of or in the course of employment." Such an injury is compensable if it causes incapacity and takes away a worker's ability to earn wages.

No one is better qualified to decide the diagnosis, the differential data and the etiologic factor than the dermatologist. When specialists disagree, an impartial and duly qualified dermatologist is appointed to examine and report, and his report is considered admissible as evidence, provided that the employee and the insurer have been seasonably furnished with copies of it (General Laws of Massachusetts, Chapter 152, Section 9). The employee who is unable to employ a dermatologist always has the right to an impartial examination.[86]

On October 14, 1935, the Massachusetts Legislature adopted an amendment to permit a claim of industrial disease to be referred to three impartial physicians, who are allowed to examine the patient, investigate working conditions as they deem necessary, and report to the Industrial Accident Board their diagnosis and opinion regarding the cause and extent of incapacity, if any. The report must be made by a majority vote of the referees. The diagnosis should be positive — that is, it should specify the disease or condition found or state specifically that no disease was found. This report is binding and final, but the Board can base its decision on the previous history or on subsequent facts. The diagnosis of the medical referees must be made from the physical signs present at the time of examination, although this diagnosis may be made months after the onset, perhaps when the patient has recovered. As a result, interesting complications have developed. The constitutionality of this statute has been extensively debated. There is no doubt

that it has prevented an employee from having a complete or full trial on his claim of industrial disease when the Industrial Accident Board appoints the three physicians. On the other hand, he is saved the expense of employing one or more experts to testify in a questionable case.[3]

In April, 1946, the provisions of the Workmen's Compensation Act that provide for the appointment of three medical referees who hold *ex parte* investigations and make reports binding on the Industrial Accident Board were declared unconstitutional by the full bench of the Massachusetts Supreme Judicial Court. The decision, written by Justice James J. Ronan, declared that the 1935 law, amended in 1938, "deprives a party of his fundamental rights" under both state and federal constitutions.

The question of cancer of the skin arising from a single injury is a cause of frequent legal and medical disputes. Such claims as the following are constantly appearing. A thirty-four-year-old man was struck by a splinter from an emery wheel, which penetrated his cheek. Probing for the splinter led to considerable trauma, followed by swelling and suppuration. The swelling became larger and harder. Two months later it measured 1.5 by 2.0 cm. and appeared as a sharply defined lesion with a verrucous center, with no pain. Removal and histologic examination revealed a prickle-cell carcinoma.[87]

Perhaps owing to the employment of older workers during the manpower shortage, one of us (J. G. D.) has seen a large increase in the number of workers making claims for skin cancer arising from a single injury or an industrial exposure. Many of these claims present evidence of a slight blow that has received first-aid treatment. Most blows have been on the face, the frequent site of cutaneous cancer in old age. Careful examination and the history of the original injury would have shown that a cancer was already there and that the trauma had caused bleeding at a site where an intact skin would have shown no evidence of injury. This sequence of injury, continued treatment and final recognition of a cancerous growth, together with inexpert or biased medical testimony, presents facts that few industrial commissioners have the temerity to dismiss.

Whether or not a fatal or disabling cancer was caused by an industrial accident is certainly a question not of law but of fact. If reasonable evidence has been introduced in the proceedings before an industrial board to sustain a finding that an industrial injury, single or multiple, has caused a cancer that resulted in disability or death and the Board so decides, an appellate court in any state in which the general rule laid down above is in force cannot set aside the award or remand the case for further proceedings, even though the court believes that the weight of all the evidence did not justify the decision. In any event, so long as the cause or causes of cancer are not known, so long as there is an opinion among the medical profession at large to the effect that a single trauma may cause cancer, and so long as physicians are willing to testify under oath that in their professional opinion the disability can and did occur in the case under consideration, the industrial commission cannot be blamed if it sometimes accepts such evidence and grants awards accordingly. In forming his opinion in a given case, the physician should bear in mind the lack of definite knowledge regarding the relation of a single injury to the causation of cancer.

Whether human cancer can be caused by a single accidental injury remains an open question; the results of clinical observations are not conclusive. Although authentic cases are recorded in which cancer arose at the site of injury, the possibility that it was under way before the injury or developed independently of it cannot be excluded. That cancer can and does arise independently of injury and that single injuries become associated with cancer in one way or another in extremely few cases cannot be denied. So far as is known, cancer in animals has not been produced experimentally by a single mechanical injury.[88] Stewart[89] states: "As far as I know, I have never seen a cancer which I could logically and irrevocably assign to single trauma and, even more important than that, I do not know that anyone has observed the development of a process which could be called 'precancerous' after a trauma."

Treatment

There is nothing new to be said about treatment, but it is necessary to reiterate that the use of antibiotics, such as penicillin, or of new bactericidal or fungicidal chemicals has no place in the treatment of uncomplicated industrial dermatoses. Despite the fact that some workers become immune to their contacts, there is no question that when a worker suffers an occupational dermatitis, the sooner he is removed from his work the speedier his recovery will be. The physician should use only soothing remedies, such as wet dressings, lotions and bland ointments.[90] A valuable new contribution, however, is the use of BAL (British anti-lewisite) in the treatment of cases of chronic arsenical and mercurial poisoning; certain physicians have also been trying it on other industrial cases.[91]

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NEW HAMPSHIRE MEDICAL SOCIETY

PROCEEDINGS OF THE ONE HUNDRED AND FIFTY-FIFTH ANNIVERSARY

House of Delegates, May 13 and 14, 1946 (Concluded)

Dr. Sycamore then assumed the chair, while Dr. Smith presented his report as delegate to the House of Delegates of the American Medical Association.

The 1945 session of the American Medical Association was curtailed to a meeting of the House of Delegates, which was held in Chicago on December 3, 4 and 5. Your delegate served on the Reference Committee on Reports of Officers. To expedite the work of the Association and express its policies more promptly, it was voted to hold two meetings of the House of Delegates each year. It was decided that the next session would be held in July, 1946, in San Francisco, and plans were developed for the centennial meeting at Atlantic City in 1947. You are all urged to attend this momentous meeting.

Dr. George R. Minot, of Boston, was awarded the Distinguished Service Award, and Dr. Harrison H. Shoulders, of Nashville, Tennessee, was elected president-elect. The Section on General Practice of Medicine was established. Dr. Roger I. Lee delivered two excellent addresses, which have been published in the *Journal of the American Medical Association*, emphasizing the democracy of the House of Delegates. County medical societies through the state societies and their delegates bring items of business and express their opinions at the meetings. Perhaps our society has been lax in this respect, for your delegate has been instructed only once during the years that he has served.

The sudden end of hostilities and the rapid demobilization of medical officers created such a demand for even the increased number of residencies that, unfortunately, some physicians were unable to obtain them. The same conditions, together with the continuation of the accelerated program in the medical schools, prevented the inauguration of as complete a plan of postgraduate instruction as had been anticipated. You are referred to the *Journal of the American Medical Association* of December 22, 1945, for the details of the excellent work of the Committee on Postwar Medical Service.

President Kretschmer delivered a practical address in which he emphasized the importance of the county medical societies. Since most of the secretaries of the county societies are present, the changes that he proposed will be stated briefly: scientific meetings of a group of counties, lasting all day and evening and thus ensuring a large gathering, better speakers and a better program; regular meetings devoted to a discussion of ethics, economics and organization; and meetings at which timely educational topics could be presented to a lay audience, and also meetings in association with the social service, relief, political, public health and similar groups.

Major General Paul R. Hawley, acting surgeon general of the Veterans Administration, told about the reorganization of his department so as to attract young physicians, to utilize part-time men in the hospitals, and to provide for the free choice of a physician by a veteran when he needs outpatient care for service-connected disabilities.

The House disapproved the Maternal and Child Welfare Act of 1945, which extends and greatly expands the E. M. I. C. program and defines a child as anyone under the age of twenty-one.

It was decided to make permanent the Bureau of Information and to urge each state society to establish a similar information service:

> This state information service should collect from various public and private agencies data relating to medical facilities, medical personnel or medical needs and other information concerning medical care within the state. This information service should at all times be in a position to furnish information concerning areas in need of physicians and a complete picture of the medical facilities, physical and economic aspects of any community within the state.

Your delegate, having done much of this work during the last five years, knows that there is a definite need in New Hampshire for such a service.

The Committee on National Emergency Medical Service was established, and has suggested that each state society appoint a similar committee. The committee will study the many communications that have been received and the suggestions made by physicians in the armed forces, and will also formulate policies for recommendations to be forwarded through the Surgeon General to the Secretary of War and the Secretary of the Navy expressing the views of the medical profession in planning for proper utilization of the services of physicians in any national emergency. Your delegate recommends the establishment of a similar committee of three civilian physicians who have served with the armed forces.

The Council on Medical Service and Public Relations and its Washington office have done a great amount of excellent work. The Board of Trustees and this Council were instructed to proceed as promptly as possible with the development of a specific national health program, with emphasis on the nationwide organization of locally administered prepayment medical plans sponsored by medical societies. In accordance with these instructions the following steps have been taken: establishment of standards of acceptance for medical-care plans that have the approval of the Council on Medical Service of the American Medical Association; organization of Associated Medical-Care Plans, Incorporated, to establish co-ordination and reciprocity among all these plans, to undertake research, to provide consultation and information services and to engage in a great campaign of public education; establishment of a division of prepayment medical-care plans to administer the activities related to the promotion and development of medical-care plans in all the states; and restatement of the national-health program of the American Medical Association.

There was considerable discussion of President Truman's national-health program and the Wagner–Murray–Dingell Bill. Official disapproval of Section 4 of the bill, which provides for compulsory health insurance, was expressed for the following reasons:

1. The Wagner–Murray–Dingell Bill is founded on the false assumption that solution of the medical care problem for the American people is the panacea for all the troubles of the needy.
2. This is the first step in a plan for general socialization not only of the medical profession but of all professions, industry, business and labor.
3. Positive proof exists from experience in other countries that inferior medical service results from compulsory health insurance.
4. A program such as outlined is enormously expensive. It will result in greatly increased taxes for the entire population of the United States.
5. Voluntary prepayment medical plans now in operation in many parts of the United States and which are rapidly increasing in number will accomplish all the objects of this bill with far less expense to the people and under these plans the public will receive the highest type of medical care.

DEERING G. SMITH

Dr. Lewin, for the Committee on Officers' Reports, recommended that the House of Delegates establish a new standing committee, the New Hampshire Emergency Committee, consisting of three doctors formerly of the armed forces. This motion was duly seconded.

Dr. Dye stated that if this were to be a standing committee the by-laws would have to be amended. The Secretary asked what the committee was to do. Dr. Smith answered that the committee would try to find out what was wrong with the medical organization in the last war and prepare for the next war. A great many complaints and a great many criticisms, some constructive, had been received from medical officers and others who had served in the war. A number of the complaints had been turned over to this Committee of the American Medical Association, and it had accordingly been recommended that each state find other constructive items of criticism that could be used in the future.

Dr. Dye suggested that the Committee on Officers' Reports, together with Dr. Smith, draw up an amendment to be submitted for approval on the following day, in such a form that the name and duties of the committee would be specified in the by-laws.

Dr. Smith stated the work of the committee could probably be accomplished in a year. It was to be like the Procurement and Assignment Service. All the criticisms that have arisen because of the participation of doctors in the war could be brought out, and constructive criticisms could be offered to the central committee of the American Medical Association, which, in turn, could be forwarded to the Secretary of War and the Secretary of the Navy, who were looking for criticisms.

The Secretary asked whether the Committee on Medical Preparedness could function in such a way. Dr. Smith replied that the committee was to be composed of veterans.

The Secretary then inquired if a special committee for one year could take care of the situation. Dr. Smith answered that it could.

Dr. Lewin asked if the American Medical Association intended to have a national committee that would be permanent. Dr. Smith did not believe so.

Dr. Lewin, for the Committee on Officers' Reports, then moved that the House of Delegates create a committee, the New Hampshire Emergency Medical Service Committee, consisting of three doctors formerly of the armed forces during the war, to be elected by the House of Delegates for a period of one year or until the next annual meeting of the House of Delegates.

Dr. Forsberg suggested that the motion be amended so that the committee would consist of five doctors. Dr. Lewin accepted the amendment. This motion was duly seconded and was carried.

Dr. Lewin stated the Committee on Officers' Reports had come to no conclusion on the question of establishment of a bureau of information, but requested open discussion by the delegates.

The Speaker said that during the last year, he had received at least fifty letters and had interviewed over thirty-five physicians regarding moving to New Hampshire. Some of these men had visited New Hampshire. They wanted to know the best locations and what industries were to be found. He said that they were looking for residencies in all types of towns, in the various specialties. He had received letters addressed to the State Planning Commission, the State Board of Health, Dr. Metcalf and Dartmouth College Medical School, all of which had been forwarded to him for reply. He believed that in the near future there would be many such requests, especially from men who had gone into the service directly from medical school. The whole effort had been to place these men in towns where no returning physician would be deprived of his practice, and also to attract them to the smaller towns and smaller areas, where there were no doctors. It had sometimes been difficult to interest these men in going to such areas. Dr. Smith believed that a central clearing house should be set up, and suggested that the Secretary continue the work in the future, although he was already overworked.

The Secretary replied that he did not come in contact with the units mentioned, and that he did not know the towns very well. He stated that Dr. Smith had been a godsend because he knew the situation at present. His only other suggestion was the State Board of Health, which received many requests for information.

The President called attention to the fact that the House of Delegates during the preceding year had created tentatively a committee for this particular purpose, with representatives from each county society. The men were to have been appointed by the county societies themselves, with a central chairman, and there would have been communication between the central chairman and the county-society members, each chosen for his temperament and his willingness and ability to serve as a local information agency from each county.

Dr. Lewin, for the Committee on Officers' Reports, recommended that the Society co-operate to the fullest extent with any private prepayment plan of medical care, as a means of warding off a compulsory health program, and moved the adoption of this portion of the report. This motion was duly seconded and was carried.

Dr. Lewin, for the Committee on Officers' Reports, then moved that the report of the delegate to the American Medical Association be accepted. This motion was duly seconded and was carried.

Dr. Smith then read the following report:

Report of the Committee on Medical Preparedness

The cessation of hostilities terminated the necessity of this committee during the past year, and it is recommended that it be discontinued.

The chairman was active in formulating requests for the return to civilian practice of physicians who were badly needed in their respective communities. He is pleased to report that a large number of physicians have considered coming to New Hampshire to practice, and many have already done so. He has answered at least fifty letters of inquiry and interviewed over thirty-five doctors. Although there are still several opportunities in the State for physicians, the areas in the greatest need are now supplied with medical care.

DEERING G. SMITH, *Chairman*
HAROLD I. L. LOVERUD
ANTHONY E. PETERS

Dr. Lewin, for the Committee on Officers Reports, moved that the Committee on Medical Preparedness be discontinued, and that the by-laws be so changed. This motion was duly seconded.

A member inquired whether all men in the service had returned to civilian life.

Dr. Smith replied that two or three doctors who had been practicing in New Hampshire before the war were still in the service.

The motion was carried.

Dr. Smith then assumed the chair.

Dr. John P. Bowler then presented the following report:

Report of the Committee on Medical Education and Hospitals

During the last year, as in previous war years, there has been a general suspension in the activities of postgraduate training, particularly along the lines of the Commonwealth Fund, which has not resumed its subsidization of postgraduate fellowships.

The New England Postgraduate Assembly will renew its program this fall. This had been successfully operating as a two-day meeting at Cambridge for several years previous to 1942, and committees are now active in preparation for the re-establishment of what was on its way to being an obviously successful venture.

The Speaker's Bureau as operated by this committee principally for the supply of Society members for county meeting programs has continued, but in lesser demand than previously. Until now, when returning men will have made the medical personnel of the State more stable, no circularizing of the membership has been done for the enrollment of volunteers as speakers. Soon this will be undertaken, and a new list accumulated.

Senate Bill 191, which provides for a survey of hospital and health facilities within the various states to provide data for later consideration of federal grants toward the costs of construction of such facilities as needed, has led to the appointment of the New Hampshire State Commission for Hospital Study. This body, which was appointed by the Governor last summer, consists of fifteen members, with Mr. James M. Langley, of Concord, as chairman. The medical representation on this commission includes Drs. George C. Wilkins and Jules Gagnon, of Manchester, Dr. Philip McQuesten, of Nashua, Dr. John P. Bowler, of Hanover, and the secretary of the Board of Health. The Governor and Council appropriated $3000 for the purposes of this commission in the carrying out of its studies.

The State Department of Health has co-operated with the commission in preparing the health-center portion of the study. The State Planning and Development Commission has provided the office quarters, and its director has served as secretary to the commission, directing the work of the field representative, Mary T. Swoboda.

JOHN P. BOWLER, *Chairman*
JAMES W. JAMESON
SAMUEL M. BROOKS

Dr. Lewin, for the Committee on Officers' Reports, recommended that the delegates from the different county societies remind their secretaries that speakers for their various county meetings are available through Dr. Bowler's committee, and moved the acceptance of this portion of the report. This motion was duly seconded and was carried.

Dr. Lewin, for the Committee on Officers' Reports, then moved that the report of the Committee on Medical Education and Hospitals be accepted. This motion was duly seconded and was carried.

Dr. John B. McKenna then presented the following report:

Report of the Committee on Mental and Social Hygiene

I must plead guilty to procrastination in the matter of the report of the committee. Undoubtedly you are aware of the fact that Dr. Baker was to have made this report, but because of his retirement and emigration from New Hampshire, he suggested to Dr. Smith that one of the other members of the committee present the report. The third member of the committee, Dr. Simon Stone, had only recently returned to Manchester, and Dr. Smith therefore asked me some time ago to do this.

To begin with I cannot do much better than forward Dr. Baker's letter to Dr. Smith, in which there are incorporated several valuable suggestions with which I am in essential agreement. My difficulty in making such a report derives from the fact that, being peripherally located, I am not in sufficient contact with the more central problems. Therefore, my remarks can be the result only of essential experiences in connection with the more peripheral problems of psychiatry and mental hygiene in the State.

My first thought in this connection is that the field of psychiatry and mental hygiene in New Hampshire is peculiarly lacking in any effective co-ordination, because there is no one person in a position to do this co-ordination, such as the Commissioner of Mental Diseases in other states. Such a person and his assistants could more directly co-ordinate any efforts toward better psychiatric care and mental hygiene. I believe that under such a person Dr. Baker's suggestion regarding the formation of a state committee on mental hygiene could operate effectively in accordance with Dr. Baker's other suggestions. To these I add the desirability and practical necessity for more widespread use of psychiatric social-service workers, who might bring into contact many otherwise neglected cases of psychiatric problems with psychiatrists and mental hygienists. It has been my experience from practically ten years of psychiatry in the northern parts of New Hampshire, that there are many cases in which earlier psychiatric intervention might have forestalled more serious consequences. This would also imply the desirability of more peripherally located mental hygiene clinics, such as those existing in other parts of the State. Such additional clinics might well be located in places like Hanover and Littleton. Again, I have no basis for any knowledge of conditions in the more eastern parts of the state.

Some time ago, in a conversation with Dr. Dolloff, I brought up for discussion the possibility that the State could employ one or more psychiatric social-service workers whose duty would be to make periodic trips through the rural parts of the State to investigate and call to the attention of psychiatrists any specific needs for psychiatric help.

There is also an undoubted need for more widespread public education in psychiatric matters. In this connection it is worthy of note that the American Psychiatric Association has formed a committee on reorganization among whose proposed plans are included definite attention to organized programs for such education throughout the country. It might be well to look into this matter so far as New Hampshire is concerned, and I shall have definite information on this point after my attendance at the American Psychiatric Conference in Chicago later this

month. At any rate, it seems obvious that the public enlightenment on psychiatric matters is a field that needs much more attention and can best be carried out by public talks throughout the State by accredited psychiatrists. This obviously implies the need for more well trained psychiatrists throughout the State. At the present time, to my knowledge, specialists in psychiatry outside Manchester and Concord are virtually nonexistent. Despite this fact there is a crying need for such specialists to share the burden of work, which is physically impossible for one or two persons. This indicates the advisability of taking some steps to attract well trained men to the State. In addition to the lack of mental-hygiene clinics, there is a strong need for facilities for the care of the mentally ill in the outlying parts of the State. It is well known that the State Hospital at Concord has been for many years tremendously handicapped by the fact that it is the only institution of its kind. Eventually some expansion of this institution or the erection of similar institutions in other parts of the State will be imperative. In the meantime, however, would it not be possible to establish, at one or more strategic points, modified institutions for the care and treatment of selected benign cases of mental disorder? In some such way the ponderous work of the present State Hospital could be materially reduced.

All these matters will necessarily require quite substantial financial expenditure. Whether or not this can all be taken care of by appropriations by the State is a matter with which I am quite unfamiliar. However, I should like to point out that the United States Government has before its consideration a bill providing for grants in aid to individuals, institutions and states throughout the country for the betterment of psychiatry in general. This should be looked into thoroughly, for I believe that this bill is likely to be passed and that New Hampshire is certainly one of the states in need of such assistance as might be afforded.

In summary, therefore, I believe that the primary need is for a co-ordinator in all psychiatric and mental-hygiene matters in the person of a director or commissioner of mental health, and that from such a department all the pertinent factors could be directed and co-ordinated and any relations with the United States Government could be established.

It is regrettable that this report must be in the nature of a theoretical discussion and not based on any statistical figures, but I believe that my distance from the central problems accounts for this lack. A further suggestion that might overcome such a deficiency is that future chairmen of the New Hampshire Committee on Mental Hygiene might be in more direct and more frequent contact with all the members of the committee, and that all might make a more active and statistical investigation of the requirements for satisfactory progress in this field.

JOHN B. MCKENNA

Dr. McKenna then read Dr. Baker's letter of March 6, 1946, to Dr. Smith, as follows:

Dear Dr. Smith:

Yes! I am guilty. I resigned and retired and arrived here in San Antonio, Texas, on December 20, 1945. I sincerely hoped not to overlook my obligations but this matter being out of sight was also out of mind. I do not expect to return to New Hampshire before June 1. I do not have the addresses of my associates on the Committee or would write them at this late hour. Suggest you or Lewin write them of the situation and ask one of them to substitute for me.

My personal feelings are that we should get in touch with the National Committee for Mental Hygiene through its medical director, Dr. George D. Stephenson, 1790 Broadway, New York City, and get suggestions for forming a good strong committee for mental hygiene in New Hampshire. If this committee had an active president and secretary-treasurer, and many active influential members willing to pay dues, it would be an agency for great good in New Hampshire. Perhaps it could include the present Social Hygiene Society. This committee could advise and co-ordinate public clinics and help support and influence public attitudes on mental-health topics.

Then all state institutions should do what a small institution some years ago, the Laconia State School, did not have the courage or vision to do, put an item in the state institution budget to cover costs of mental-hygiene clinics.

Clinics cannot be run for nothing. I think Dr. Dolloff had funds left to that institution during the days of the Bancrofts, father and son. Massachusetts has many institutions. All these state institutions carry a heading for mental hygiene in their budgets, and the legislature allows it. If New Hampshire Medical Society approved such a course, I am sure it would be a strong influence with the Committee on Appropriations.

I am sorry to be so lame in this matter, but with the approval of the other members of my committee, I recommend (1) that a New Hampshire Committee on Mental Hygiene be formed; (2) that George Stephenson be written to for assistance; (3) that this committee supervise and co-ordinate mental-hygiene activities in New Hampshire and educate the public on this subject; and (4) that all state institutions for mental defect, disease and derangement, including the industrial school and state's prison, put an item in their budgets for extramural mental-hygiene work.

Sincerely,
BENJAMIN W. BAKER

Dr. Lewin, for the Committee on Officers' Reports, moved that the Committee on Mental Hygiene take the lead in this field of hygiene clinics, in educating both the public and the lawmakers. This motion was duly seconded.

Dr. Clough inquired if the members of the committee could not provide, during the year to follow, a definite motion or program to be presented to the Governor of New Hampshire for funds to be raised for psychiatric clinics.

The President pointed out that there was great need in the State for outpatient psychiatric care and patient care for the people unable to meet the tremendous expense of the facilities available for private care. He therefore wished to hear from the members of the committee regarding a concrete plan for the State.

Dr. Lewin, for the Committee on Officers' Reports, accepted the suggestion that the matter be turned back to the committee for a definite report at the next meeting. This was seconded, and the motion was carried.

The report of the Committee on OPA Assistance was then presented, as follows:

Report of the Committee on OPA Assistance

The District Food-Rationing Officer of the OPA called on your committee for advice regarding the need for additional unrationed foods in some hundreds of cases. As restrictions were removed on the sale first of processed foods and then of meats, these requests became less numerous. With the discontinuance of rationing, which we hope may come soon, this function of your committee will be at an end.

In deciding on the merits of so many applications, it has of course been necessary to be somewhat dogmatic. We have followed in the main the recommendations of the Subcommittee on Medical Food Requirements of the National Research Council, but we have tried to give a liberal interpretation.

Your committee early lost one member through removal to another state, and a second member was on duty outside New Hampshire during much of the year. This, together with the more urgent problems caused by rationing, prevented any effective consideration of the overall nutritional situation in New Hampshire. However, your chairman did discuss these matters with Miss Hinman, the state nutritionist, on two or three occasions.

In more normal times, which we hope are at hand, such a committee could be of definite value in stimulating interest in good nutrition throughout the State.

There is as much "hidden hunger" in New Hampshire, especially in some rural areas, as in most other parts of the country. Stressing these problems and the best means of dealing with them, discussing them before the state and county medical society meetings and before nursing groups, as well as giving the state nutritionist every encouragement, should in the end be productive of definite results.

COLIN C. STEWART, *Chairman*
SIMON STONE

Dr. Lewin, for the Committee on Officers' Reports, moved that this report be accepted and

that the Committee on OPA Assistance be continued until the Government discontinues OPA through Congressional action. This motion was duly seconded and was carried.

The report of the Committee on Public Health was presented by the chairman, Dr. Harris E. Powers:

The Committee on Public Health started out the year with the following members: Dr. Harris E. Powers, chairman, Dr. Alfred Frechette, and Dr. Anthony E. Peters. During the course of the year, however, we had the misfortune of losing Dr. Frechette, who accepted a call out of the State. New Hampshire and the New Hampshire Medical Society lost a very valuable man through this transfer.

In the previous year your committee had the pleasure of inspecting some of the lumber camps in the northern sections of the State, and this year conducted an inspection tour of the Portsmouth area, which has been one of the more concentrated industrial areas owing to the vast expanse in the Navy Yard during the war. On July 25, 1945, Dr. Frechette, Dr. McCloud, of the State Health Department, and your chairman proceeded to Portsmouth, where we met Dr. Peters. An inspection was conducted of one of the large milk plants in Portsmouth, and the method of handling milk from its entry into the plant, the processing of the milk and finally the bottling and preparation for delivery were covered. The pasteurizing of milk and the by-products, such as cottage cheese and ice cream, were followed through by your committee. The progress in the handling of milk in the past twenty years has been remarkable, but there is still room for improvement through the education of employees along sanitary lines. These efforts are being conscientiously carried out by the state health authorities.

From Portsmouth we followed along the South Shore to Seabrook, where the clam industry flourishes. Few people in the State realize the magnitude of this industry, and the small town of Seabrook seems to be the center of this activity. Scattered over the town are small shacks, which are used by the shuckers who receive the clams as they are dug and remove the meat from the shell. These individual industries are all small and few of them have anything like modern equipment. Of the many shacks that we inspected only one had stainless-steel benches; all the rest had wooden trays or benches. The process of shucking consists of removing the clams from the shells — this is all done by hand and with the aid of a short chisel-like knife; each clam is handled separately, the shell being cast in one pile and the clams being tossed in a container of one-gallon content. It was amazing to find that many of the clams had been dug from remote sections in Massachusetts and Maine, shipped to Seabrook in large barrels, shucked, and then transported back to wholesalers throughout New England. This is probably accounted for by the fact that the shuckers have established themselves in this community over a period of many years. They are a more or less isolated group, intermarrying generation after generation. The income of these employees seems adequate, and several of the workers said that they averaged six or seven dollars a day at their work. Their houses are extremely primitive, if the average laborer's house is used as a standard, and their customs and habits are equally singular. If their houses may be used as a guide they seem to have little interest in the niceties of life. The largest plant that we inspected employed ten to twelve people, the average being about six to ten employees. The State Health Department inspects these plants regularly for sanitary conditions and endeavors to improve the standards of the industry by screens on the windows, cleanliness in the shuckers themselves and boiling water and steam for sterilization purposes.

After inspecting the Seabrook area we proceeded to the Hampton Beach area. Some of the fire hazards were inspected and suggestions made; the proximity of the buildings at Hampton Beach result in a great fire hazard, and it is hoped that sufficient equipment will be retained at the beach to prevent such a catastrophe.

There is a great need in the communities that we inspected for education of both employers and employees along sanitary lines, particularly regarding handling of food supplies. It is certainly impossible for the Health Department to cover these territories as frequently as seems necessary with the small staff of inspectors available. It is one thing to make a suggestion and quite another to follow this suggestion up by routine calls to see that it is carried out. If sufficient inspectors were available to the Health Department to conduct these routine tours much more rapid progress and much better sanitary conditions would prevail.

Your committee has had no special problems turned over to it during the last year. We have endeavored to keep abreast with the work of other public health committees. It seems possible that, from time to time during the course of the year, your committee will conduct inspection tours of the various industries in the State similar to the one just described and will thereby keep a more active contact with the Health Department and industry throughout the State, submitting its report annually to the Society.

HARRIS E. POWERS, *Chairman*
ALFRED FRECHETTE
ANTHONY E. PETERS

Dr. Lewin stated that during the national emergency, owing to the great shortage of labor, the practice of sanitation in restaurants, hotels, food stores and industries had been allowed to become alarmingly lax, and that Dr. Powers had repeatedly referred to the shortage of public-health inspectors. Dr. Lewin moved that the House of Delegates go on record as stating that the sanitation in the restaurants, hotels and the food handling industries of the State are lax. He also moved that the public-health laws of the State be more rigidly enforced, and that the Secretary send a note to the Health Department and to the Governor to that effect. This motion was duly seconded by Dr. Sycamore.

The secretary inquired how effective such a step would be. Dr. Wilkins replied that such action would probably do little good unless the Legislature could be induced to appropriate more money to hire more inspectors. The city boards of health did not have enough money to conduct all the inspections required by law. The remedy was up to the Legislature rather than to the Governor or the State Board of Health.

The President stated that it might be worth while for the delegates to pass a resolution voicing the opinion of the Society that larger appropriations were needed to make the proper personnel carry out the provisions already in the law.

Dr. Sycamore asked if the matter should be referred to the Committee on Public Health. The President answered that the decision could be left to the discretion of the Secretary, who could send a note to the proper authorities. He moved that the Secretary send a note to the proper authorities. This motion was duly seconded and was carried.

Dr. Lewin, for the Committee on Officers' Reports, moved that the Report of the Committee on Public Health be accepted.

This motion was duly seconded and was carried.

The report of the Committee on Tuberculosis was then presented by Dr. Robert B. Kerr.

The report of the Committee on Tuberculosis was presented, as follows:

Report of the Committee on Tuberculosis

Since the appointment of this committee last May one of its members, Dr. Rufus R. Little, formerly superintendent of the New Hampshire State Sanatorium, has accepted the position of superintendent of the Bergen County Hospital at Ridgewood, New Jersey. During his service in New Hampshire Dr. Little made an outstanding contribution to the success of the program for the cure and prevention of tuberculosis. As medical director of the New Hampshire State Sanatorium he greatly expanded the service in chest surgery. This program had been ably inaugurated by his predecessor, the late Dr. Robert M. Deming.

Your committee is happy to report that the State has emerged from World War II without evident injury to the health of its people. The death rate from all causes has decreased to a low point. The tuberculosis death rate, which increased slightly in the year 1943, was reduced to an all-time low — 18.7 per 100,000 population — in 1945. This rate is one of the lowest among the states of the Union.

It is an axiom accepted by vital statisticians that the trend in death rates from infectious diseases should be evaluated on the basis of analysis by three-year periods. Study of the tuberculosis mortality statistics on this basis demonstrates that the rate has been maintained on a downward basis for the last thirty years, during which an increasingly intensive campaign has been waged against the disease by the medical profession, public-health workers, the state government and the general public.

Further analysis of the tuberculosis mortality statistics by three-year periods discloses that the reductions in the early years of the intensive attack against the disease were large. As the mortality has declined these reductions have decreased, even with the increasing effectiveness of activities for the eradication of the disease. The average annual tuberculosis death rate for the three-year period 1916–1918 was 117.2 per 100,000 population, and in the succeeding period 1919–1921 was 92.7. This represented an aggregate reduction of 25 per 100,000 population. Again studying the three-year period 1940–1942 we see that the annual average tuberculosis death rate was 26.6. In the three-year period 1943–1945 the rate was 23.3. This is a reduction of 3.3. This analysis demonstrates a well recognized fact, acknowledged by vital statisticians and students of communicable diseases, namely, that it requires even more intensive effort to reduce a low tuberculosis death rate than a high one. The irreducible minimum in the death rate from tuberculosis is conjectural. Some public-health authorities estimate it as a rate of 15 per 100,000 population. If the present effective campaign against the disease is continued we should reach this rate by 1954 (based on an annual average rate for the three-year period at that time). The medical profession, however, has adopted as its goal the virtual eradication of the tubercle bacillus, and will press on to that end.

Many members of the profession and workers in the public-health field feared that the onset of the greatest war in human history would bring about a reversal of the reductions in the tuberculosis mortality. This reversal has not occurred. It may be said that several factors contributed to the continued decline in the death rate during the war years, despite longer hours of labor for the people and the strains and stresses, both physical and mental, under which they worked.

First, the population did not have to undergo serious malnutrition or exposure to debilitating diseases. Second, the medical and public-health resources of the State functioned at a high level of efficiency. These resources were greatly augmented by the tremendous increase in chest x-ray surveys conducted for selective-service registrants in the State Induction Center, in the state-wide program of the New Hampshire Tuberculosis Association in its chest diagnostic clinics and in its schools and college tuberculin-test surveys, and in the joint program of the State Board of Health and the State Tuberculosis Association of chest x-ray films for workers in industry.

During the last five years more than 80,000 chest x-ray films have been taken in these various surveys, in addition to the large number taken in hospitals throughout the State by request of members of the New Hampshire Medical Society.

So much has been said in this report and in previous reports regarding the encouraging reductions in the tuberculosis death rate that it might be thought that the disease has been brought to a point of minor significance in the lives of the people of the State. This is far from the truth, and such an impression might lead to relaxation of the intensive campaign that has been waged against this disease. Such relaxation of effort would inevitably lead to disastrous results, and the ancient enemy, the tubercle bacillus, would again increase its ravages among the people.

The tragic fact remains that even today tuberculosis is the master thief of human lives and efficiency caused by infectious diseases in the State. The tubercle bacillus is still unconquered and a major cause of disease and death.

In the summer of 1945 one of the members of your committee, aided by the Division of Vital Statistics of the State Board of Health, prepared for general distribution a booklet entitled "Tuberculosis Facts and Figures for New Hampshire," which presented a series of tables and graphs illustrating the status of the tuberculosis problem.

Table VIII in the series is entitled "Tuberculosis — The Leading Cause of Death from Communicable Diseases in New Hampshire for the Past Fifteen Years by Five-Year Periods." The chart portrays the tragic fact that in the five-year period 1940–1944 tuberculosis caused 621 deaths, whereas the following number of deaths were caused by other infectious diseases in the same period: whooping cough, 45; infantile paralysis, 23; measles, 18; scarlet fever, 16; typhoid fever, 7; diphtheria, 2; and smallpox, 2. Nor does this table tell the complete story of the ravages of the disease, which is not one of short duration, but an infectious malady disabling the afflicted for many months and often years whether the outcome is cure or death. The vital statistics tell only of deaths. They do not portray the valiant services of the sanatoriums or the courage and sacrifice on the part of the patients and their physicians and nurses that have resulted in the cure and healing of tuberculosis among thousands of New Hampshire's people.

Two years ago one of the members of your Committee made a study of the diagnoses among persons admitted to the chest diagnostic clinics of the New Hampshire Tuberculosis Association. It was found that the physicians of New Hampshire referred to these clinics for final diagnosis the group of people in whom were discovered the largest number of active cases of pulmonary tuberculosis.

Ten per cent of all patients referred to the clinics that year by the physicians had active cases of pulmonary tuberculosis. One hundred and eighty-five out of the 390 practicing physicians in the State referred patients to the clinic.

There is no doubt that the most important of all case-finding agencies in the fight against tuberculosis in the State are its practicing physicians. It is almost always true that the family physician has the first opportunity not only to ascertain the presence of tuberculosis among the people, but also to give battle for the cure of the afflicted and to safeguard the other members of the family from the tubercle bacillus. For it is to the family physician to whom most people go when troubled by signs of ill health.

The records in the chest diagnosis clinics prove that the physicians of the State, if they are determined to do so, can perform a better job of suspecting and discovering active tuberculosis cases, year in and year out, than any other agency.

In the same study of the population of chest diagnostic clinics it was found that the second largest number of significant cases of tuberculosis was found among the contacts. For the most part these persons were referred to the clinics by the tuberculosis nurses, with the approval of the family physicians.

What of the future? There is no preventive for tuberculosis such as medical science has provided in toxoid for diphtheria and vaccine for smallpox, nor is there any specific cure.

We must carry on and intensify our present program of cure and prevention, utilizing to the fullest extent the resources that have already proved to be so effective.

As has already been outlined in this report, we have arrived at the point of diminishing returns for efforts expended in the tuberculosis program — that is, we must

work harder to be sure of continued reductions in the death rate. We appeal to the members of the Society for their continued aid and co-operation. We urge better and prompter reporting of tuberculosis cases by the practicing physicians to the State Board of Health. (The report slip states that the reports are confidential.) We suggest that the members of the Society request aid when it is needed in the examination of contacts and so forth. We urge further consideration of the practicability of routine chest x-ray examination of patients admitted to general hospitals. In this connection your committee hopes that in the coming year a plan may be worked out for such a study in one or two general hospitals in the State. The findings may be indicative of similar samplings elsewhere and may give more definite information regarding the possible values of such a procedure in New Hampshire.

ROBERT B. KERR, *Chairman*
RICHARD C. BATT

Dr. Lewin referred to a point emphasized by Dr. Kerr — the advisability of routine chest x-ray study of all patients admitted to general hospitals, but stated that the committee had recently consulted x-ray men and learned that film is still hard to obtain, so that the carrying out of this suggestion for the present seemed impracticable. He added that when x-ray film again became plentiful, however, one or two hospitals in the State should try routine chest x-ray examinations for a year, and the results should be reported at a future meeting of the House of Delegates.

Dr. Lewin, for the Committee on Officers' Reports, moved that the House of Delegates go on record as favoring a one-year trial of routine chest x-ray study by two hospitals in the State when x-ray film again became plentiful. This motion was duly seconded.

Dr. Sycamore stated that no difficulty in obtaining film had been experienced in his section of the State. He believed that the proposed study would be interesting, but that the question of its economic desirability would depend on the amount of unsuspected disease uncovered by such a survey.

The President asked who would be responsible for the cost of the study.

Dr. Dye inquired what hospitals would be selected and by whom.

Dr. Sycamore replied that these matters could be worked out by the New Hampshire Tuberculosis Association.

Dr. Johnston stated that several Boston hospitals had carried out surveys, which had proved satisfactory.

Dr. Clough expressed his belief that the State Hospital could carry out the study.

Dr. Sycamore said that the State Hospital was a special type and that perhaps its study would not have as much value as one conducted by a general hospital.

The speaker asked who was to pay for the study.

Dr. Lewin answered that Dr. Kerr probably believed that the Tuberculosis Association would have something to do with the expense of the study.

A member stated that the Society should approve the general adoption of the study, rather than stipulate a trial of approving something that has adequately been proved in other places and thus not contribute anything more than what has been contributed in other places.

Dr. Lewin replied that the mention of two hospitals was merely for the purpose of obtaining a report on the experience of those institutions.

Dr. Parsons pointed out that some patients routinely admitted to a hospital paid $10.00 for a chest x-ray film, but that if the State took the x-ray film for $1.00, the $10.00 could not be collected.

Dr. Biron suggested that, instead of enumerating the number of hospitals, the Society recommend that routine studies be done wherever possible.

The President stated that routine studies should be performed on everybody in the State, and that he believed that the Society should approve in principle universal x-ray studies. He did not, however, believe that the survey should be limited to ordinary patients admitted to general hospitals.

The motion was carried.

Dr. Amsden then presented the following report:

Report of the Necrologist

The following members of the New Hampshire Medical Society have died since the last report of the necrologist:

NAME	ADDRESS	DATE OF DEATH
Abbott, Clifton S.	Laconia	December 11, 1945
Bates, John H.	East Rochester	March 17, 1946
Brown, Lester R.	Laconia	November 1, 1945
Cutler, Charles H.	Peterboro	December 22, 1945
Foster, George S.	Manchester	July 11, 1945
Hildreth, Lewis G.	Marlboro	June 25, 1945
Hoyt, Park R.	Laconia	August 31, 1945
Joyce, Roland J.	Nashua	July 8, 1945
McLaughlin, Patrick J.	Nashua	September 8, 1945
Powers, John E.	Wilton	July 16, 1945
Sanders, Loren A.	Concord	April 24, 1946
Scott, Nathaniel H.	Wolfeboro	January 25, 1946
Smith, Henry O.	Hudson	May 14, 1945
Tarbell, Wallace H.	Contoocook	July 20, 1945
Taylor, Herbert L.	Portsmouth	October 24, 1945
Wallace, Ellen A.	Concord	November 2, 1945
Wilder, Richard E.	Whitefield	August 24, 1945

HENRY H. AMSDEN

Dr. Lewin for the Committee on Officers' Reports, moved that the report of the Necrologist be accepted. This motion was duly seconded and was carried.

The speaker expressed the Society's thanks to Dr. Lewin and his committee for their excellent work. He then asked whether the delegates approved a request from the New Hampshire Department of Public Welfare for the appointment of Dr. John A. Coyle to the Medical Advisory Committee of Ophthalmologists.

The Secretary stated that the Department of Public Welfare had a group of six or seven ophthalmologists with whom they consulted regarding cases of blindness, and that the department selected the men it wished to serve on the committee. As a matter of courtesy, they had always asked the Society to ratify or approve the appointments.

Dr. Biron moved the appointment of Dr. J. Coyle to the committee. This motion was duly seconded and was carried.

Dr. Sycamore stated that he had been instructed by the Grafton County Medical Society to ask that Drs. Percy Bartlett and Elmer H. Carleton be made life members, and moved that they be made life members. This motion was duly seconded and was carried.

The President read the following letter.

March 26, 1946

Dr. Richard W. Robinson, President
New Hampshire Medical Society
79 Lincoln Street
Laconia, New Hampshire

Dear Dr. Robinson:

At two recent meetings, the New Hampshire Roentgen Ray Society, which represents all the radiologists in this state, unanimously reaffirmed that x-ray service in hospitals is a professional, not a hospital service. We are particularly opposed to having diagnostic radiology included as a hospital service in Blue Cross policies, because this creates a discriminatory barrier between the radiologist and the other members of the profession. Radiology requires the services of a medical specialist and therefore represents medical practice. Since hospitals are not permitted to engage in medical practice, they cannot legally sell medical radiologic service to an insurance company. No useful purpose can be served by separating the radiologist from the profession and demoting him to the rank of a nonmedical hospital employee.

We believe that the present situation, if allowed to continue, will result in radiologists' abandoning hospital work and confining their activities to private office practice. This would lower the high standards of hospital service and medical care which the New Hampshire Medical Society and the New Hampshire Roentgen Ray Society attempt to maintain.

Upon request of the New Hampshire Roentgen Ray Society, I would, therefore, ask you to give me an opportunity to explain our position to the House of Delegates at the next meeting.

Sincerely yours,

(Signed) Fred S. Eveleth, M.D., *President*
New Hampshire Roentgen Ray Society

Be It Resolved, That the House of Delegates of the New Hampshire Medical Society hereby expresses itself as opposed in principle to any policy whereby the professional services of any doctor of medicine be charged for and dispensed by any other agent than the doctor concerned, and that the House of Delegates is, therefore, specifically opposed to the present system whereby, in violation of their charters, hospitals dispense for fee the services of roentgenologists and pathologists.

The President added that in the report of Dr. Smith, as delegate to the American Medical Association in 1939, as presented to the House of Delegates, it was definitely stated that the American Medical Association had gone on record as taking the same position, and that the only permissible basis on which a hospital could utilize this service was that of a payment of money to a patient, with which the patient could recompense the doctor. He moved the adoption of the resolution. This motion was duly seconded by Dr. Clough and others.

Dr. Sycamore observed that the directors of the Blue Cross recognize the validity of this position, their only question being whether it was practical, because of the fact that radiologists were in hospitals, and patients considered x-ray study a hospital service. He believed that co-operation among the Blue Shield, the Blue Cross and radiologists would settle the issue.

A member stated that if the proposed change were made, the Enabling Act for selling insurance would result in the transfer of physicians to the Blue Shield from the Blue Cross, since participants of Blue Cross were not allowed to sell professional services of any description.

Dr. Biron recalled that at a recent meeting of the New Hampshire Pathological Society this subject had been discussed in a similar manner and that a similar resolution had been adopted. The Blue Cross contract had also taken up some of the diagnostic work in neurology, in which the situation was analogous to that in the laboratory and the x-ray room; the Blue Cross was considering the matter.

The motion was carried.

Dr. Parsons then stated that the welfare patient rates in Coos County should be increased.

A member remarked that he had been approached by the members of a county society regarding the fee of $20.00, which included prenatal care and delivery and which had not been changed since the war, and that he had been asked to inquire what was being done elsewhere — for example, in the large cities.

The President said that he was sure that the Department of Public Welfare was concerned about the situation and that it would like to meet the proper recompense in welfare cases. Two years previously the fee for office and house calls had been definitely increased, and the department had also permitted the addition of medicine up to a cost of $1.00, without the necessity of listing the type of medicine given. The President further pointed out that many of the state societies were fumbling through the same problem, the answer to which was a provision for service through insurance, using the basic insurance rate under which patients under the plans could get medical care. The greatest hindrance resulted from the fact that the insurance plan had not been accepted throughout the State. Acceptance was necessary before the Department of Welfare shifted its own medical-care burdens onto the shoulders of physicians.

The Speaker stated that he had been informed by the Secretary that the Department of Public Welfare had increased the rates 25 per cent.

A member inquired whether such a fee included prenatal care. The speaker replied in the affirmative.

A member stated that certain welfare organizations simply referred to a fee schedule made up in the days of the depression by a committee headed by Dr. Fuller, who took into account the conditions at that time, and that some committee should revise the suggested fee schedule to conform to present conditions.

The President recommended that the Society favor the fee schedule that had become a part of the voluntary insurance plan to cover the care of the indigent of the State.

Dr. Appleton moved that a fee schedule for indigent patients be made up by the Department of Public Welfare along the lines of and on the same basis as the Blue Shield.

The Secretary stated that the Blue Shield had no fee schedule and asked if indemnity payments were meant. Dr. Appleton replied in the affirmative.

This motion was duly seconded and was carried.

A member moved that since there was no further business the meeting adjourn. This motion was duly seconded and was carried.

Whereupon, the first meeting of the House of Delegates was adjourned at 11:55 p.m.

CARLETON R. METCALF, *Secretary*

* * *

The House of Delegates reconvened at the Hotel Carpenter, Manchester, on May 14, 1946, at 9:00 a.m., with Speaker Deering G. Smith, of Nashua, presiding.

The following members answered the roll call:

The President, *ex-officio*
The Vice-President, *ex-officio*
The Secretary-Treasurer, *ex-officio*
Samuel Feiner, Ashland
Earl J. Gage, Laconia
Francis J. C. Dube, Center Ossipee
W. J. Paul Dye, Wolfeboro
Albert C. Johnston, Keene
Marjorie Parsons, Colebrook
Israel A. Dinerman, Canaan
Leslie K. Sycamore, Hanover
Robert E. Biron, Manchester
George C. Wilkins (alternate for Samuel Fraser), Manchester
Loren F. Richards, Nashua
Robert R. Rix, Manchester
Deering G. Smith, Nashua
Frank J. McQuade, Franklin
William P. Clough, Jr., New London
Philip M. L. Forsberg, Concord
Willard C. Montgomery, Epping
Samuel T. Ladd, Portsmouth
Robert W. Tower, Plaistow
William R. Latchaw, Somersworth
Raymond R. Perreault, Rochester
B. Read Lewin, Claremont

The speaker declared a quorum present.

The first order of business was the report of the Nominating Committee by Dr. Richards, who nominated for President Drs. Ralph W. Tuttle, of Wolfeboro, Edward R. B. McGee, of Berlin, and Charles W. Hannaford, of Portsmouth.

The Speaker asked if any further nominations for President were forthcoming.

Dr. Biron moved that the nominations be closed. This motion was duly seconded and was carried.

The Speaker appointed Drs. Dinerman, Gage and Feiner as tellers.

On written ballot, Dr. Ralph W. Tuttle was elected president.

Dr. Richards nominated for Vice-President Drs. Clarence Dunbar, of Manchester, Robert Holmes, of Keene, and John Hunter, of Dover.

The Speaker asked if there were any additional nominations from the floor.

Dr. Biron moved that the nominations be closed. This motion was duly seconded and was carried.

On written ballot, Dr. Hunter was elected vice-president.

A partial report of the Committee on Amendments to the Constitution and By-laws was presented by Dr. Dye, who stated that the committee moved to amend Section 11 of Chapter VIII of the by-laws to create a new standing committee, to be known as the Council of the New England State Medical Societies, consisting of three members.

This motion was duly seconded by Dr. Biron.

The speaker stated that the title of the committee could not be the Council of the New England Medical Societies.

Dr. Dye expressed his willingness to have the title changed.

The Speaker stated that representatives to the council could be chosen.

Dr. Sycamore pointed out that the representatives were delegates to the council.

Dr. Wilkins asked what the former name of the group had been. The President replied that the title was given in old records as "New England Council."

The Speaker stated that the present name was the Council of the New England State Medical Societies and that the council could not be created at the meeting. The members from the Society could be called representatives to the council, delegates to the council or members of the council. He suggested the word "delegates."

The Secretary observed that the title had formerly been the "New England Medical Council" but had been changed to the "Council of the New England State Medical Societies." He preferred to list the delegates as members of a standing committee, as the Committee on Medical Hygiene was listed, with the heading, "Council of the New England State Medical Societies."

The motion, as amended, was carried.

Dr. Dye moved that Section 11, Chapter VIII, of the by-laws be amended to read as follows:

> The Committee on the Control of Cancer shall consist of five members whose duty it shall be to study conditions in this state and bring to the attention of the Society and of the general public such matters concerning the prevalence, prevention and cure of the disease as may seem to them advisable.

This motion was duly seconded and was carried.

The President then moved that the Secretary cast one ballot for the election of the remainder of the slate of candidates. This motion was duly seconded and was carried.

The Secretary announced that he had cast the ballot, electing the officers and committees as named on the slate.

The Speaker then declared the officers and committees elected to their respective offices for the ensuing year.

Dr. Dye, for the Committee on Amendments to the Constitution and By-laws, recommended that the proper section of Chapter VIII of the by-laws be amended to discontinue the Committee on Medical Preparedness, and moved the adoption of this

portion of the report. This motion was duly seconded and was carried.

Dr. Dye then asked the delegates to approve a change, which would subsequently be read before the open meeting, in Article IV, Section 1, of the Constitution to read as follows: "*Section 1.* This society shall consist of members, life members and honorary members." He also asked that Article IV, Section 3, be amended to read as follows: "*Section 3.* Life members shall be those members whose dues are remitted."

Dr. Biron moved these changes in the Constitution be approved. This motion was duly seconded and was carried.

Dr. Dye moved that Chapter X, Section 1, of the by-laws be amended to read as follows: "An assessment of $10.00 per capita on the membership of the component societies is hereby made the annual dues of this society."

This motion was duly seconded by Dr. Dube and was carried.

Dr. Sycamore, stating that it was the duty of the House of Delegates to vote in approval of the Board of Directors of the New Hampshire Physicians' Service, moved that the House approve the following list of directors: Harry L. Additon, Monsignor Buckley, O. E. Cain, Francis J. C. Dube, Fred Fernald, James W. Jameson, James M. Langley, Joseph E. LaRochelle, Carleton R. Metcalf, J. J. Morin, Ray W. Pert, Richard W. Robinson, C. A. Rollins, James Ross, Frank J. Sulloway, Joseph Winter and L. K. Sycamore.

This motion was duly seconded and was carried.

The President expressed gratitude to the Manchester Medical Society for their arrangements of this meeting, and moved a vote of thanks.

This motion was duly seconded and was carried.

The Speaker asked if there were any further business.

Dr. Biron moved that the meeting be adjourned. This motion was duly seconded and was carried.

The second and final meeting of the House of Delegates was adjourned at 9:45 a.m.

CARLETON R. METCALF, *Secretary*

CASE RECORDS OF THE MASSACHUSETTS GENERAL HOSPITAL

Weekly Clinicopathological Exercises

FOUNDED BY RICHARD C. CABOT

TRACY B. MALLORY, M.D., *Editor*

BENJAMIN CASTLEMAN, M.D., *Associate Editor*

EDITH E. PARRIS, *Assistant Editor*

CASE 32391

PRESENTATION OF CASE

A sixty-two-year-old forest consultant entered the hospital because of marked weakness, loss of weight and burning pain over the buttocks and dorsolumbar region.

Six months before entry, having previously been in excellent health, the patient noted a sense of fullness in the upper abdomen and put himself on mild dietary restrictions to lose weight. Not long after this he noted that he continued to lose weight even though he no longer observed the diet and had a good appetite. In the six months before entry he lost 15 pounds. Five months before entry he noted excessive fatigue after minor physical exertion and on several occasions felt completely exhausted after an ordinary day's work. This fatigability steadily increased until he confined himself to his house and grounds and finally to his room and bed. Three months before entry all the teeth had been extracted. A few days later the patient had chills and fever and generalized malaise and felt completely exhausted for ten days. A physician discovered that the liver was enlarged, the edge being palpated three fingerbreadths below the costal margin.

Examination of the blood at another hospital revealed a red-cell count of 3,900,000, with 12.4 gm. of hemoglobin, and a white-cell count of 2350, with 30 per cent neutrophils, 67 per cent lymphocytes, 1 per cent monocytes and 2 per cent eosinophils. There was poikilocytosis, anisocytosis and polychromatophilia of the red cells. The icteric index was 8. The nonprotein nitrogen was 25 mg., the cholesterol 200 mg., the cholesterol esters 174 mg. and the total protein 7 gm. per 100 cc., with 4.3 gm. of albumin and 2.7 gm. of globulin. The alkaline phosphatase was 2.5 Bodansky units. A heterophil antibody reaction was negative. The stools were negative for blood. X-ray films of the chest, lumbar spine and pelvis, a gastrointestinal series and an intravenous pyelogram were all reported normal. A plain film of the abdomen suggested some enlargement of the liver and spleen. A barium enema revealed what appeared to be a mild diverticulitis in the sigmoid area. Cystoscopy showed a normal prostate and bladder.

The patient complained of a burning sensation in the feet and over the buttocks. He was given 2 cc. of Lederle's concentrated liver extract twice a week and then once a week, with gradual increase in the red-cell count and hemoglobin to 4,500,000 and 13 gm. respectively. The white-cell count rose as high as 5600 and the percentage of neutrophils

went up at one point to 60 per cent. The patient, however, grew steadily weaker and had more trouble with burning pain in the buttocks and back, although the burning sensation in the feet disappeared. The appetite began to fail, and he had great difficulty in sleeping because of nervousness and the burning sensation. Weakness became so marked that he confined himself to his room. Some fibrillation in the muscles was noted. Although the patient was afebrile, occasional sweats occurred, and he felt warmer than usual. He was finally admitted to this hospital for study.

Physical examination revealed a nervous, depressed, markedly weakened man, with a flushed face and neck. The thyroid gland was palpable but not enlarged. The heart and lungs were normal. The liver edge was felt four fingerbreadths below the costal margin. The spleen was not palpable. There was an area of hyperesthesia over the left buttock. The legs were somewhat weak, with questionable atrophy about the knees.

The temperature was 98°F., the pulse 120, and the respirations 20. The blood pressure was 130 systolic, 85 diastolic.

Examination of the blood revealed a red-cell count of 4,000,000, with a hemoglobin of 70 per cent, and a white-cell count of 2500, with 11 per cent segmented and 20 per cent band neutrophils, 3 per cent eosinophils, 42 per cent small lymphocytes, 12 per cent large lymphocytes, 1 per cent immature lymphocytes, 1 per cent monocytes and 5 per cent blast cells of the white series. The red cells varied in size and showed some polychromasia. Several normoblasts and stippled cells were present. The alkaline phosphatase was 14.4 Bodansky units, the serum phosphorus was 4.2 mg., the cholesterol 154 mg., the cholesterol esters 97 mg., the nonprotein nitrogen 23 mg. and the total protein 6.84 gm. per 100 cc., with 4.4 gm. of albumin and 2.4 gm. of globulin. The van den Bergh test was normal. The bromsulfalein test showed 50 per cent retention of the dye. The prothrombin time was normal. The urine was normal, showing no bile. Several determinations of the basal metabolic rate ranged from +41 to +23 per cent.

X-ray studies of the chest and pelvis were normal. A plain film of the abdomen showed a normal liver and suggestively large spleen.

The patient spent most of his time in bed because of weakness. The pulse ranged between 100 (while the patient was sleeping) and 120. Frequent injections of Demerol were required to relieve the burning pain in the back and buttocks.

On the fifth hospital day a liver biopsy was performed.

Differential Diagnosis

Dr. Wyman Richardson: Dr. Castleman was good enough to give me a blood smear to examine, which I did before I looked at the record. This blood smear showed abnormal white cells, to which I shall return; a total white-cell count that was less than normal, with a relative but not absolute increase of lymphocytes; polymorphonuclear neutrophils, what there were of them, showing definite toxic granules; reduced platelets, in conjunction with the decrease in neutrophils; and occasional heavy polychromatophilic and nucleated red cells.

What can one say of such a blood smear? There was red-cell regeneration, as shown by the nucleated red cells and the polychromatophilic cells. What conditions cause such regeneration? Hemolytic anemia might, but hemolytic anemia and reduced platelets do not occur in the same patient. In other words, hemolytic anemias are associated with leukocytosis and an increase of platelets. Red-cell regeneration follows acute blood loss, in which, again, one finds increased platelets. Red-cell regeneration occurs in bone-marrow encroachment. In such cases the white-cell count and the platelets vary, depending on the cause of encroachment. That brings us back to the abnormal white cells. In the record they are given as 5 per cent. The blood smear disclosed 5 per cent or more immature cells, which I could not identify and which may be called stem cells. That percentage seemed high enough to be significant. There were also some necrotic white cells that, if they had been of the same type, would have increased the percentage; there is no way of determining the type.

The blood picture was quite typical of acute leukemia. Do you want me to discuss the case further?

Dr. Benjamin Castleman: That is up to you.

Dr. Richardson: I shall just run through the protocol.

The reason for extracting the teeth is not indicated in the record.

The chief symptoms in the history were increasing fatigue and a curious type of burning pain over the buttocks and dorsolumbar region that suggests some sort of nerve-root pain.

The description of the blood is consistent with my findings on examination of the blood smear.

I presume that the heterophil reaction was tested in this case in a search for a benign disease. I am not sure whether the barium enema showed diverticulitis or diverticulosis. There is nothing in the record to suggest active inflammatory diverticulitis.

There was no improvement from liver therapy, because the red-cell count was within the limit of error and both the two red-cell counts and the two hemoglobin determinations were comparable, unless done by a photoelectric-cell technic. Therefore, the patient did not respond to treatment on the basis of the evidence given in the record, but he apparently did not lose ground.

Fibrillation in the muscles does not mean a great deal to me. I do not believe that some type of neuromuscular disease need be considered; such a condition occurs with a loss of considerable weight and tissue of any type.

It seems probable that a four-hour chart would have disclosed that the patient actually had fever.

It is always difficult, for me at least, to make an adequate study of sensation, even when involvement of the nerves and nerve roots is known, and it is frequently difficult to demonstrate a definite sensory change. Perhaps the hyperesthesia over the buttock can be regarded as definite evidence of nerve-root irritation, which the history suggests.

I interpret all the laboratory tests, except for the alkaline phosphatase, as normal. The result of the bromsulfalein test suggests involvement of the liver. I assume that the liver and spleen were enlarged. It is difficult to settle this point by x-ray examination; in fact, I am not at all sure that even the pathologist can settle it. I suppose when one weighs any organ one still does not know how much it weighed during life, that is, whether it was heavier or lighter. I can conceive of a spleen that could be shrunken or filled up.

It seems to me that a bone-marrow biopsy was indicated and probably would have given as much if not more information than a liver biopsy.

I have very little to add to my original diagnosis. The blood picture is consistent with that of some disease encroaching on the marrow. The clinical findings suggest anemia and weakness and what appears possibly to have been nerve-root pain. Aside from the findings in the blood, the laboratory tests were essentially negative, except for the increased phosphatase and the bromsulfalein retention. There was possibly an enlarged liver, about which the clinicians and the roentgenologist disagreed to some extent. It seems to me that the clinical diagnosis fitted a subacute or acute — although I should call it subacute — aleukemic leukemia, which is my diagnosis. That means that the liver had presumably been invaded. Invasion of the liver by lymphoma occurs quite frequently, late in the disease, but primary invasion with primary symptoms is unusual. I remember a case with severe changes in which the disease seemed to be largely confined to the liver, with such a rapid downhill course that the patient was believed to have acute degenerative disease of the liver. Such a case is unusual, however, and I should say that the patient in the case under discussion had lymph nodes along the paravertebral areas, with possible involvement inside the spinal canal and with pressure on nerve roots, and that he may also have had a diffuse lymphocytic infiltration of the liver. One might also say that there was no disease of the liver, but the results of the bromsulfalein test remain to be explained. The alkaline phosphatase might be accounted for on the basis of disease involving the bone, with osteoblastic activity, or on the basis of liver disease, most frequently seen in biliary obstruction but perhaps occurring oftener in neoplasm than any other condition. I have found, however, that the alkaline phosphatase is not always dependable. I disregarded it in the last case that I discussed — much to my discomfiture, because the patient turned out to have malignant tumor with biliary obstruction.

The basal metabolic rate makes one think of thyroid disease. The basal metabolic rate is elevated in both leukemia and aleukemic leukemia. The age of the patient, the way in which the disease progressed and the nerve-root symptoms allow me to make a guess regarding the clinical diagnosis — subacute aleukemic leukemia, probably lymphoblastic. Pathological examination probably disclosed a lymphoblastic lymphoma.

DR. LAURENCE L. ROBBINS: This point on the difference of opinion regarding the size of the liver and spleen should be straightened out. I interpreted these films, and although I knew that the liver was enlarged clinically, I could not say that it was enlarged from the films. That is not an infrequent discrepancy. The size of the liver in the x-ray films is not too accurate, whereas demonstration of the size of the spleen is much more accurate. So long as the clinical findings are indicative I should not be disturbed if enlargement of the liver did not appear in the x-ray films.

DR. RICHARDSON: The liver is often only two or three fingerbreadths below the costal margin, but no mention is made of the upper level of dullness. Frequently the liver is in a low position and is not enlarged. I believe that four fingerbreadths indicates enlargement, and I accept the statement that the liver was enlarged; there is no statement, however, about the upper limit of dullness.

DR. JOHN GRAHAM: The upper limit of dullness, as I remember, was below the fifth rib.

DR. RICHARDSON: Then the liver was enlarged.

DR. N. M. MARTIN: Should you consider the possibility of a reticulum-cell tumor, as opposed to the lymphoblastic type?

DR. RICHARDSON: Yes; only I do not believe that the stem cells or immature cells would have appeared in the blood film.

DR. MARTIN: They might, if there had been a large amount of spread.

DR. RICHARDSON: In such cases the abnormal cells are monocytes, or an actual monocytic leukemia is present. I cannot tell from this blood picture whether or not the cells were monocytes, which usually occur in patients of middle age.

DR. EARLE M. CHAPMAN: Is a low white-cell count and lymphocytosis, in the presence of infiltration in the liver of other cells than monocytic cells, possible in leukemia? Such findings are seen in Chiari's disease, in which there are focal areas of liver degeneration.

DR. RICHARDSON: There are not usually many abnormal cells in the blood. Any patient with a large congested spleen is likely to have a leukopenia, which is often seen in cirrhosis when there is an enlarged spleen. I should not say that actual liver involvement is necessary.

DR. GRAHAM: I saw this patient just a week before admission, when the suggestive symptoms were weakness, loss of muscular strength and painful cutaneous sensations, the most prominent symptoms being the severe pain in the back and the burning sensation in the buttocks that kept him awake all night, despite every attempt to find a sedative to relieve him. The impressive point in the physical examination before admission was the constantly rapid pulse (120), with a normal temperature. Hyperthyroidism might have been considered, because of a lid lag, a flushed face and neck and a rapid irregular pulse. The patient lay in bed in a cool house with only a sheet over him, but he claimed that he was warmer than anyone else in the house. For over three months he was known to have had an enlarged liver. Smears performed at his home failed to reveal any abnormal cells; those done when he first consulted a physician showed a low white-cell count, with lymphocytosis. The basal metabolic rate was +41 per cent. On admission there was a question of changes in the liver resulting from some form of thyrotoxicosis, cirrhosis, carcinoma of the liver or leukemia. Pernicious anemia seemed to be ruled out when there was no response to therapy. I observed the abnormal cells in the smear and inclined toward a diagnosis of leukemia. A biopsy was done on the liver rather than the bone marrow chiefly because it was easier and was the one organ that appeared to be pathologically affected.

DR. CHAPMAN: Was the patient an alcoholic?

DR. GRAHAM: No. He took an occasional drink but was by no means an alcoholic.

CLINICAL DIAGNOSIS

Aleukemic leukemia.

DR. RICHARDSON'S DIAGNOSIS

Subacute aleukemic leukemia (lymphoblastic lymphoma).

ANATOMICAL DIAGNOSIS

Aleukemic leukemia (?monocytic or lymphatic), with massive liver involvement.

PATHOLOGICAL DISCUSSION

DR. CASTLEMAN: An aspiration biopsy taken by Dr. Wade Volwiler was quite satisfactory. Diffusely infiltrating the liver, more marked in the sinuses, were large abnormal cells that I could not identify with certainty, but that were certainly leukemic cells. I thought that cells of the myeloid series could be ruled out. The cells were typical of either the monocytic or the lymphoid type; I rather favored the former, since many cells had horseshoe-shaped nuclei that were more in keeping with that diagnosis. Dr. Graham showed the slide to Dr. Frederic Parker, Jr., pathologist at the Boston City Hospital, who was also in favor of the monocytic group.

Dr. Graham, will you tell us what happened to the patient after the biopsy?

DR. GRAHAM: I cannot give a final answer. It was decided to give some x-ray therapy starting over the back, in the hope of relieving the burning pain in the back if it was due to nerve-root infiltration. After the third or fourth treatment, as I remember it, the patient developed jaundice and became extremely sick, so that the benefit from x-ray therapy was regarded as not worth the attending discomfiture. In fact the patient decided that for himself.

I should like to ask Dr. Richardson what his experience has been regarding the relation of the basal metabolic level and leukemia. Is there any connection with the white-cell count or any other findings?

DR. RICHARDSON: Several people have used the basal metabolic rate as a basis for deciding on the necessity of x-ray treatment. The test is difficult to do accurately. It seems to me that one can get along about as well with one's own clinical findings, such as fever, malaise and the blood picture.

CASE 32392

PRESENTATION OF CASE

First admission. A twenty-seven-year-old housewife entered the hospital because of urinary drainage from an operative wound.

The patient's health had been excellent until fifteen months before entry, when she noticed crampy pains in the lower abdomen that began shortly after a menstrual period and gradually increased in severity. She was taken to another hospital, where she was told that she had pelvic trouble and given intensive treatment with penicillin and sulfonamides, to some of which she was sensitive. After a month of this regime a laparotomy revealed bilateral tubal abscesses, with leakage of pus from the left tube. The tips of both ovaries, which were likewise diseased, were resected along with the tubes. Following the operation, the patient noted that when she was given an enema there was leakage from the suprapubic wound, usually consisting of purulent material, but that fecal matter seemed to be extruded when the bowels were loose. Two months before entry a second operation, which was performed to remove the sinus tract, revealed that the bladder was also involved; postoperatively, urine, as well as fecal material, drained from the suprapubic wound. The drainage of urine was particularly profuse when natural voiding was delayed. Although the appetite had returned somewhat, at the time of admission the patient weighed 80 pounds, as compared to her usual 123 pounds. She also had difficulty with postprandial gas and constipation.

Since the onset of her illness she had lost a considerable quantity of hair, and much of what remained had turned gray. The patient had been married five months before she was taken ill. There was nothing in the history to indicate venereal exposure.

Physical examination revealed a poorly nourished woman in no acute discomfort. The heart and lungs were normal. The abdomen showed a palpable nontender mass low in the left lower quadrant. The old midline scar appeared well healed, but along the recent low suprapubic incision there were two draining sinus tracts surrounded by granulation tissue. There was slight clubbing of the fingers and toes.

The temperature was 99.5°F., the pulse 95, and the respirations 20. The blood pressure was 120 systolic, 75 diastolic.

Examination of the blood revealed a hemoglobin of 10.0 gm. and a white-cell count of 21,400, with 79 per cent neutrophils and 17 per cent lymphocytes. The total protein was 5.7 gm. per 100 cc., with 3.3 gm. of albumin and 2.4 gm. of globulin. The nonprotein nitrogen was 15 mg., the sugar 90 mg., the calcium 8.8 mg. and the phosphorus 4.1 mg. per 100 cc. The carbon dioxide was 25.8 milliequiv. per liter.

The urine showed a +++ test for albumin, with a few red cells and 250 white cells in clumps per high-power field. Repeated cultures grew colon bacilli and nonhemolytic streptococci.

An intravenous pyelogram revealed slight embarrassment to drainage on the left side but no other abnormality of the upper urinary tract. There was some irregularity of the left side of the bladder, and dye seemed to be escaping, from either the bladder or the left lower ureter, into the surrounding soft tissue and possibly into the bowel. An x-ray film of the chest revealed no evidence of active parenchymal disease, but a few prominent markings in the left first interspace were suggestive of old scars. A barium enema was not entirely satisfactory because of the patient's inability to retain enough barium to fill more than the rectum. The bowel above the level of the sigmoid was widely filled with retained gas and fecal material. At the level of the rectosigmoid, barium escaped into a sinus tract, the destination of which could not be determined.

On the ninth hospital day a transverse colostomy was performed. The patient recovered uneventfully.

Second admission (four months later). The patient showed little or no improvement after discharge. Drainage continued from the sigmoid fistula, producing a thin purulent material rather than feces.

Physical examination and laboratory findings were essentially unchanged from those of the previous admission. On the fifth hospital day an operation was performed.

Differential Diagnosis

Dr. Richard Chute: This previously healthy woman had been married for five months when she started to have crampy lower abdominal pains beginning shortly after a catamenial period and increasing in severity so that she was taken to another hospital where she was given a month's intensive treatment with penicillin and sulfonamides, apparently with no improvement, since she had to be operated on. At operation bilateral tubal abscesses were present, with leakage of pus from the left tube. The tips of both ovaries were also diseased and were resected along with the tubes.

Although tuberculosis is the cause of only 5 per cent of all cases of salpingitis, that disease seems likely in this case, since the process was acute, with pus draining from at least one tube, even after a month's intensive treatment with penicillin and sulfonamides. This treatment would have been enough to improve greatly any salpingitis due to the gonococcus and would probably have made an operation unnecessary. Also, there was nothing in the history to indicate venereal exposure. Other evidence of tuberculosis is that x-ray examination of the chest revealed old healed tuberculosis. Although these x-ray findings were not extensive, there was clubbing of the fingers, suggesting extensive chronic pulmonary disease.

After operation a fistula draining sometimes pus and sometimes feces developed and persisted for more than a year. The fact that it persisted so long without healing suggests that it was due to a chronic disease with little tendency to heal, to a partial obstruction of the intestine distal to the fistula or to the intermittent filling up and spontaneous drainage of an abscess connecting with the bowel, such as might occur with diverticulitis of the sigmoid. The patient was rather young for diverticulitis, but tuberculosis could well account for this condition. If the tubes and ovaries were tuberculous and one tube was leaking frank pus, a tuberculous peritonitis with bowels matted with dense adhesions may have occurred. In such a situation a fecal fistula could easily have been caused accidentally during surgery, or a tuberculous lymph node may have broken down, with abscess formation and a fecal fistula that was kept open by partial blockage of the intestine by a tuberculous peritonitis. Nothing is said about sulfur granules to suggest actinomycosis, and I would not expect endometriosis to give this picture, especially starting from a salpingitis.

Two months before entry an operation not only failed to cure the fecal fistula but also created a urinary fistula. The record states that the bladder was involved, but whether by adhesions or by some active disease is not indicated. One can well imagine that the pelvis was a tangled mass of dense adhesions about a purulent fecal draining sinus, with destruction of all normal anatomy, and that a urinary fistula was accidentally created during surgery. The two months' duration of the fistula was rather long for involvement of the bladder, unless some obstruction to urination existed, which is unusual in a

woman, or unless tuberculosis of the urinary tract were present, which I am inclined to believe was not the case. The urine showed a +++ test for albumin, with a few red cells and many white cells per high-power field, but the source and therefore the significance of the white cells in the urine is in doubt, since it is not stated whether or not this was a catheterized specimen. It is well known that in women the voided urine may contain many white cells from the vagina whereas a catheterized specimen from the same person may show no white cells. Also, in the presence of a urinary fistula, some white cells and a few red cells would probably have been present, even in a catheterized specimen. The repeated urine cultures showing a mixed infection with colon bacilli and nonhemolytic streptococci are also somewhat against urinary tuberculosis, in which the urine frequently gives a negative culture for the ordinary organisms. Furthermore, there was no history of dysuria or frequency, which usually occurs in urinary tuberculosis, and the intravenous pyelograms were normal. Therefore, I am inclined to believe that tuberculosis of the urinary tract did not exist. In contradistinction to bladder fistulas, those arising from the lower ureter often persist for a long time. On the basis of the x-ray film showing irregularity of the left side of the bladder, slight embarrassment to drainage of the left ureter and escape of the dye from the bladder or the left lower ureter into the surrounding soft tissue, and also because of the history that the drainage of urine was particularly profuse when natural voiding was delayed, I suspect that in the region of the left ureterovesical junction there was an opening not only into the bladder but also into the lower part of the left ureter.

On admission there was evidence of marked weight loss, anemia, a slight fever and a white-cell count elevated to 21,000. I can find nothing in the laboratory studies that does not fit in with this picture of cachexia and infection or that is of any particular significance in the differential diagnosis. The patient was having difficulty with postprandial gas and constipation, which is consistent with the diagnosis of tuberculous peritonitis. A nontender mass was palpable in the left lower quadrant. This, with the loss of weight and anemia, naturally raises the question of cancer. I prefer to explain the patient's clinical findings on the basis of one disease, however, and therefore guess that this mass was inflammatory and tuberculous.

Following the transverse colostomy the fistula, which the history revealed to be in the sigmoid, did not heal in four months. This may well have been due to obstruction distal to the fistula in the sigmoid caused by the tuberculous inflammation and mass. In fact, obstruction in this area was suggested by the x-ray report that the bowel above the level of the sigmoid was widely filled with retained gas and fecal material.

In conclusion, my diagnoses are healed pulmonary tuberculosis, tuberculosis of both fallopian tubes and of both ovaries, tuberculous peritonitis, with fecal fistula of the sigmoid, urinary fistula in the region of the left ureterovesical junction, and nontuberculous cystitis.

Clinical Diagnosis

Vesicosigmoidal cutaneous fistula.

Dr. Chute's Diagnoses

Tuberculous salpingo-oophoritis, bilateral.
Tuberculous peritonitis, with sigmoidal fistula.
Urinary fistula: left ureterovesical junction.
Cystitis, nontuberculous.
Pulmonary tuberculosis, healed.

Anatomical Diagnoses

Tuberculous salpingo-oophoritis.
Tuberculous peritonitis, pelvic.
Vesicosigmoidal cutaneous fistula.

Pathological Discussion

Dr. Benjamin Castleman: At operation the surgeon, Dr. Robert R. Linton, found the bladder, uterus, adnexa, sigmoid, cecum and appendix fairly well matted together. The rest of the abdominal cavity was free from adhesions. There was a large granulating area in the left side of the pelvis that extended from the suprapubic sinus tract through the broad ligament and to the side of the sigmoid, where a fistula was demonstrated. The left tubo-ovarian mass was removed, and the fistula repaired. An abscess containing about 50 cc. of pus was encountered in the right broad ligament close to the appendix. The abscess was drained, and the appendix removed. This raised the question whether the whole process had originated in the appendix. A suprapubic tube was inserted into the bladder, and the wound closed.

Microscopic sections of the tubo-ovarian mass and serosa of the appendix showed active tuberculosis. Postoperatively the patient continued to drain a small amount of pus from the suprapubic wound and was transferred to a sanatorium.

Dr. Robert R. Linton: In my opinion this case illustrates one of the tragedies of surgical treatment of pelvic inflammation, as well as the importance of obtaining material for microscopic examination to establish a diagnosis. When I first saw this patient I did not even think of tuberculosis. I must commend Dr. Chute on his excellent discussion of this case, which he analyzed almost perfectly. I had assumed that the vesicosigmoidal skin fistula, which had resulted from the two previous operations carried out at another hospital, was due to an error in surgical technic. The purpose of the defunctioning transverse colostomy was, of course, to divert the fecal current so that a definitive type of operation could be carried out in the pelvis after the

inflammation had subsided. The patient did not improve so much as I had hoped following this operation, but it seemed advisable to proceed with an attempt to close the sigmoid and vesical fistulas. At operation on the pelvis I was unable to visualize anything in the gross that suggested tuberculosis; there was so much secondary infection in the inflamed and ulcerating areas that there was nothing diagnostic of tuberculosis. I made what I thought was a satisfactory closure of the sigmoid fistula, but as follow-up barium enema studies showed, there was still a fistulous tract connecting the pelvis with the sigmoid. This may have been due to the fact that my suture line did not stay closed, or possibly there was another fistula that I did not find.

In view of the x-ray evidence of the persistent fistula it was obvious that the transverse colostomy could not be closed at that time. It was considered advisable to send the patient to a sanatorium in an attempt to build up her physical condition and give the tuberculous process a chance to quiet down. It was hoped that at a later date, after the pelvic inflammatory condition had become arrested, it would be possible to close the transverse colostomy. It may be necessary, however, before doing this to do a complete hysterectomy, with removal of the other tube and ovary. Despite the fact that I did not make a diagnosis of tuberculous salpingitis I do not believe that the plan of treatment would have been changed very much if the diagnosis had been made prior to my second operation. Radical removal of the uterus and adnexa would have been an extremely hazardous procedure in view of the marked secondary infection due to the connection of the sigmoid and vesical fistulas with the pelvis.

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ETHER CENTENARY

The *Journal* has previously commented on this, the year 1946, as marking the close of a century since anesthesia was introduced to the world as a necessary prelude to major surgery, the demonstration having been made at the Massachusetts General Hospital. Elsewhere in this issue may be found a complete program of the centennial exercises. A century of medical history has given its verdict that the distinction of first appreciating the significance of surgical anesthesia is peculiarly the property of the Massachusetts General Hospital, and it is appropriate that the hospital's outstanding tradition should be based on this discovery.

The occasion of the Ether Centenary is a particularly appropriate one, moreover, at which to present the immediate financial needs of the hospital to the public that it has served so well. These needs are four, comprising a fund to provide a building for the Massachusetts General Hospital Institute of Medical Science, the Massachusetts General Hospital Research Fund to implement this institute, the General Fund to provide for the innumerable expenses of the hospital's operation and the McLean Research Fund to enable the Scientific Research Department of McLean Hospital to further its research in the field of brain diseases and their cure.

The following quotation from an address reprinted in the October 14, 1815, issue of the *Columbian Centinel* seems pertinent at this juncture:

> It is not intended to solicit donations by private application to individuals. All good citizens who shall read the address will feel it their duty to contribute in proportion to their ability and will require no further solicitation. The public attention is earnestly called to the facts contained in this address.

And who would be left unmoved by the final paragraph of the address?

To the People of the Commonwealth of Massachusetts

Fellow Citizens:

. . . . Besides the Undersigned are willing to confess, that they are not ambitious of being the guardians of a charity merely nominal; they are satisfied that the sum, affixed by the Legislature as the condition of its grant, is so small, when compared with the wealth of individuals and the greatness of the state, that no plea, arising from "the hardship of the times," "the general embarrassment of affairs" or "claims of other charities," can or ought avail the community. If such a proposal as this fail, it will be in the judgment of the undersigned, decisive of the fate of the establishment. It will then be apparent, that the will is wanting in the public, to patronize such an undertaking; and that the honor of laying the foundation of a fabric of charity, so noble and majestic, must be left for times, when a higher cast of character predominates, and to a more enlightened and sympathetic race of men.

T. H. Perkins **Tristram Barnard**
Josiah Quincy **George G. Lee**
Daniel Sargent **Francis C. Lowell**
S. Higginson Jun **Joseph May**
Richard Sullivan **Joseph Tilden**
Gamaliel Bradford

Boston, 8th January, 1814.

HOSPITAL SURVEY AND CONSTRUCTION ACT

ORGANIZED medicine will in general welcome the provisions of the Hospital Survey and Construction Act, recently signed by President Truman, as indicated by the support of such organizations as the American Medical Association and the American Hospital Association during the period when the legislation was under consideration in the Senate as the Hill–Burton Bill. For the first time in American history, a balanced and inclusive program providing for a better distribution and an improved quality of hospitals has been established, and this program involves no undue extension of governmental control to private practice.

The act authorizes the appropriation of $375,000,000 for the construction of hospitals and health centers during the next five years, as well as $3,000,000 for state-conducted surveys of need. The share of each state in the appropriation for the survey and the construction program will be determined on the basis of population and per-capita income, with the qualification that federal funds may not exceed a third of the total cost of any project. The state must present to the Surgeon General of the United States Public Health Service a plan for construction based on a survey conducted by a single state agency and must appoint a properly qualified advisory council to consult with that agency. Approval of any project depends on sufficient evidence that two thirds of the total cost of construction is available from other than federal sources and that financial support is adequate to maintain and operate the institution after completion. The state is granted latitude to develop and administer its program under standards specified by the Surgeon General and the Federal Hospital Council, which consists of eight members appointed by the Federal Security Administration.

Some of the shortcomings noted by Goldmann[1] in his cogent analysis of the Hill–Burton Bill remain in the act, but there is some question whether these disadvantages are not blessings in disguise. Thus, requirements concerning standards of operation of the hospitals to be constructed are not enumerated, the fees to be charged by participating physicians are not specified, and the amount of the appropriation appears small in view of immediate needs for additions and replacements of hospital buildings. The problem involved is as old as Aesop's King Log: how much — or how little — government is best for the governed. As pointed out in a previous issue of the *Journal*,[2] the inadequacies of the distribution of medical care are a legitimate concern of government, but the nature of medical standards and the fees to be charged for medical services are matters that are better left to the medical profession; the appropriation, although small in comparison with the billions usually associated with federal grants and agencies, should prove sufficient, when augmented by state funds, to provide for the construction envisaged by the act. These shortcomings offer a challenge and an opportunity to physicians, who by individual and concerted action can render federal definitions, regulations and immense appropriations unnecessary.

REFERENCES

1. Goldmann, F. Health bills pending in Congress: part three. *New Eng. J. Med.* 234:727-731, 1946.
2. Editorial. Inadequacies of medical care. III. Those concerning its distribution and cost. *New Eng. J. Med.* 234:539, 1946.

MASSACHUSETTS DEPARTMENT OF PUBLIC HEALTH

CONSULTATION CLINICS FOR CRIPPLED CHILDREN IN MASSACHUSETTS UNDER THE PROVISIONS OF THE SOCIAL SECURITY ACT

CLINIC	DATE	CLINIC CONSULTANT
Lowell	October 4	Albert H. Brewster
Salem	October 7	Paul W. Hugenberger
Gardner (Worcester subclinic)	October 8	John W. O'Meara
Haverhill	October 9	William T. Green
Brockton	October 10	George W. Van Gorder
Greenfield	October 14	Charles L. Sturdevant
Springfield	October 15	Garry deN. Hough
Worcester	October 18	John W. O'Meara
Pittsfield	October 21	Frank A. Slowick
Hyannis	October 24	Paul L. Norton
Fall River	October 28	David S. Grice

Physicians referring new patients to clinic should get in touch with the district health officer to make appointments.

NOTICES

NEW ENGLAND DERMATOLOGICAL SOCIETY

The regular meeting of the New England Dermatological Society will be held in the Skin Outpatient Department of the Massachusetts General Hospital on Wednesday, October 9, at 2:00 p.m.

NEW ENGLAND HOSPITAL FOR WOMEN AND CHILDREN

The monthly clinical conference and meeting of the staff of the New England Hospital for Women and Children will be held on Thursday, October 3, in the classroom of the Nurses' Residence at 7:15 p.m. There will be a discussion of unusual post-partum complications. Dr. Bernadette M. Ryder will be chairman.

THE ONE HUNDREDTH ANNIVERSARY OF THE FIRST PUBLIC DEMONSTRATION OF ETHER ANESTHESIA

October 14, 15 and 16 — Massachusetts General Hospital

The Registration Desk will be located in the Rotunda of the Moseley Building at the hospital and all who attend the meetings or the symposiums are requested to register. Information about the various meetings, luncheons and evening functions, as well as tickets, will be obtainable at this desk.

All scientific symposiums will be held at the hospital. The medical public and the general public are cordially invited to attend; no tickets are required, and admission is free.

Those wishing to attend the House-Pupil Alumni Dinner at the Copley-Plaza Hotel should obtain tickets in advance, preferably by mail on the forms that have been sent out for this purpose. These tickets can be picked up at the Registration Desk, and if advance reservations have not been made, reservations and tickets can be obtained at the Registration Desk up to 3:00 p.m. on Monday, October 14. Attendance is limited to house-pupil alumni.

Tickets for the Wives' Dinner held the same night at the Hotel Somerset may also be obtained at the Registration Desk.

Luncheon will be served each day at the hospital for all those attending the symposiums, and on Wednesday, October 16, the luncheon is, in addition, for the house-pupil alumni and their wives.

Applications for tickets to Massachusetts General Hospital Theater Night on Tuesday, October 15, may be reserved by writing to the Ether Centenary Committee at the hospital prior to October 8. These tickets can then be obtained at the Registration Desk. The medical public and all friends of the hospital are cordially invited to this theater performance.

Admission to the Sanders Theater exercises in Cambridge on Wednesday, October 16, at 8:15 p.m. is by invitation only.

A commemorative motion picture of the history of the development of ether anesthesia and methods of manufacture of ether will be shown daily at the hospital and is open to all those attending the meetings.

MONDAY MORNING, OCTOBER 14
9:30 to 12:30

Surgery

DR. EDWARD D. CHURCHILL, *Chairman*

Military Surgery in World War II. DR. MICHAEL E. DEBAKEY, New Orleans. Associate professor of surgery, Tulane University of Louisiana School of Medicine.

The Surgical Approach to the Problem of Malignant Disease. DR. CHARLES B. HUGGINS, Chicago. Professor of surgery, School of Medicine, University of Chicago.

The Development of Surgery of the Joints. DR. MARIUS N. SMITH-PETERSEN, Boston. Formerly, clinical professor of orthopedic surgery, Harvard Medical School, and chief of the Orthopedic Service, Massachusetts General Hospital.

The Present Status of Surgery of the Autonomic Nervous System in Relation to Hypertension. DR. REGINALD H. SMITHWICK, Boston. Professor of surgery, Boston University School of Medicine, and surgeon-in-chief, Massachusetts Memorial Hospitals.

MONDAY NOON, OCTOBER 14

Luncheon on the Bulfinch Lawn.

MONDAY AFTERNOON, OCTOBER 14
2:00 to 5:00

Progress in the Study of Degenerative Diseases

DR. JOSEPH C. AUB, *Chairman*

The Origin and Nature of Degenerative Joint Diseases. DR. WALTER BAUER, Boston. Associate professor of and tutor in medicine, Harvard Medical School, and physician, Massachusetts General Hospital.

Hopeful Aspects of the Future of So-Called Degenerative Cardiovascular Disease. DR. PAUL D. WHITE, Boston. Clinical professor of Medicine, Harvard Medical School, and physician, Massachusetts General Hospital.

The Aging of Bone and the Treatment Thereof. DR. FULLER ALBRIGHT, Boston. Associate professor of medicine, Harvard Medical School, and physician, Massachusetts General Hospital.

Characteristics of the Mammary Cancer Milk Agent in Mice. DR. JOHN J. BITTNER, Minneapolis. George Chase Christian Professor of Cancer Research and director, Division of Cancer Biology, Department of Physiology, University of Minnesota Medical School.

Accelerated and Retarded Aging of the Skin and Its Consequences. DR. EDMUND V. COWDRY, St. Louis. Professor of anatomy, Washington University School of Medicine, and director of research, Barnard Free Skin and Cancer Hospital.

* * *

Wives of alumni and of out-of-town guests. Sightseeing tours (inquire at Registration Desk).

MONDAY EVENING, OCTOBER 14
6:30

Massachusetts General Hospital House-Pupil Alumni Association Dinner

COPLEY-PLAZA HOTEL

Presiding: DR. ARLIE V. BOCK, *President*. Speakers: DR. EDWARD D. CHURCHILL, DR. STAFFORD WARREN AND DR. JOHN SNYDER.

Massachusetts General Hospital House-Pupil Alumni Wives' Dinner

HOTEL SOMERSET

TUESDAY MORNING, OCTOBER 15
9:30 to 12:30

The Fundamental Nature of the Anesthesia Process

DR. FRANCIS O. SCHMITT *Chairman*

The Colloidal Structure of Nerve. Dr. Francis O. Schmitt, Boston. Professor of biology, Massachusetts Institute of Technology.

The Effects of Ether and Other Anesthetics on the Electrical Properties of Nerve Fibers and Cells. Dr. R. Lorente de Nó, New York City. Member, Rockefeller Institute for Medical Research.

The Effects of Narcotics on the Activity and Oxygen Consumption of Nerve. Dr. Detlev W. Bronk, Philadelphia. Director, Johnson Foundation for Medical Physics. (With Dr. F. Brink and Dr. P. W. Davies.)

Anesthetics and Cell Metabolism. Dr. Ralph W. Gerard, Chicago. Professor of physiology, University of Chicago.

Correlations and General Remarks. Dr. Irving Langmuir, Schenectady. Nobel Laureate and associate director of research, General Electric Company.

TUESDAY NOON, OCTOBER 15

Luncheon on the Bulfinch Lawn.

TUESDAY AFTERNOON, OCTOBER 15
2:00 to 5:00

Basic Problems of Anesthesia

Dr. Henry K. Beecher, *Chairman*

Effect of Anesthetics on Chemical Mediation of Nerve Impulses. Dr. Robert D. Dripps, Philadelphia. Assistant professor of surgery (anesthesia), University of Pennsylvania School of Medicine, and director of anesthesia, Hospital of the University of Pennsylvania.

The Anesthetic Activity of Fluorinated Hydrocarbons. Dr. Benjamin H. Robbins, Nashville. Professor of anesthesia and associate professor of pharmacology, Vanderbilt University School of Medicine.

Procaine Block of the Sympathetic Nerves in the Study of Intractable Pain and Circulatory Disorders. Dr. James C. White, Boston. Assistant professor of and tutor in surgery, Harvard Medical School, and chief of the Neurosurgical Service, Massachusetts General Hospital.

Differential Spinal Block. Dr. Julia Arrowood, Boston. Instructor in anesthesia, Harvard Medical School, and assistant anesthetist, Massachusetts General Hospital. Dr. Stanley J. Sarnoff, Boston. Research fellow in surgery, Harvard Medical School, and clinical fellow in surgery, Massachusetts General Hospital.

The Effect of Anesthesia on Cardiac Output in Man. Dr. Everett I. Evans, Richmond. Associate professor of surgery, Medical College of Virginia. (With Dr. B. W. Haynes and Dr. E. Smith.)

Some Effects of Anesthesia, Load and Composition on the Excretion of Water and Salt. Dr. Carl A. Moyer, Dallas. Professor of experimental surgery, Southwestern Medical College. (With Dr. F. A. Coller, Dr. V. Iob, Dr. L. Bryant and Dr. H. Vaughan.)

Implications concerning Cellular Nutrition during Anesthesia. Dr. Henry K. Beecher, Boston. Henry I. Dorr Professor of Research in Anesthesia, Harvard Medical School, and chief of the Anesthesia Service, Massachusetts General Hospital. (With Dr. M. F. Warren and Miss A. J. Murphy.)

* * *

Wives of alumni and of out-of-town guests. Music and tea at the Isabella Stewart Gardner Museum (2:00 to 5:00 p.m.).

TUESDAY EVENING, OCTOBER 15

Massachusetts General Hospital Theater Night.

Plymouth Theater

John C. Wilson presents Noel Coward and Clifton Webb in "Present Laughter."

WEDNESDAY MORNING, OCTOBER 16
9:30 to 12:30

Physiologic Effects of Wounds

Dr. Tracy B. Mallory, *Chairman*

The Internal State of the Recently Wounded Man. Dr. Henry K. Beecher, Boston.

Blood Volume in the Wounded. Dr. Fiorindo A. Simeone, Boston. Assistant professor of surgery, Harvard Medical School, and assistant surgeon, Massachusetts General Hospital.

Blood Chemistry. Mr. Seymour Shapiro, Yonkers. Director, Biological Laboratory, Arlington Chemical Company.

Hematologic Aspects. Dr. Eugene R. Sullivan, Boston. Assistant in medicine, Massachusetts General Hospital.

The Clinical Picture of Renal Insufficiency following Wounding. Dr. Charles H. Burnett, Boston. Chief resident in medicine, Massachusetts General Hospital.

The Pathology of Shock. Dr. Tracy B. Mallory, Boston. Assistant professor of pathology, Harvard Medical School, and chief of the Department of Pathology and Bacteriology, Massachusetts General Hospital.

WEDNESDAY NOON, OCTOBER 16

Luncheon on the Bulfinch Lawn for Former Massachusetts General Hospital House Pupils and Their Wives.

WEDNESDAY AFTERNOON, OCTOBER 16
2:00 to 5:00

The Hospital in the Community

Dr. J. H. Means, *Chairman*

Ether Day: A study in public relations. Dr. Reginald Fitz, Boston. Lecturer on the history of medicine and assistant to the dean, Harvard Medical School, and consulting physician, Peter Bent Brigham Hospital.

The Voluntary Hospital — How Can It Survive in the Modern World? Dr. Nathaniel W. Faxon, Boston. Director, Massachusetts General Hospital and Massachusetts Eye and Ear Infirmary.

Role of Patients in the History of the Massachusetts General Hospital. Miss Ida M. Cannon, Boston. Formerly, chief of social service, Massachusetts General Hospital.

The Hospital: A look ahead. Dr. George R. Minot, Boston. Nobel Laureate; professor of medicine, Harvard Medical School, and head of the Harvard Department of Medicine, Boston City Hospital.

The Problem of Social Medicine: Equilibrating the distribution and technology of medical care. Mr. Winslow Carlton, New York City. Chairman of the Board of Group Health Insurance, Incorporated, New York City.

The Teaching Hospital Service to the Public. Dr. Allan M. Butler, Boston. Professor of pediatrics, Harvard Medical School, and chief of the Children's Medical Service, Massachusetts General Hospital.

5:00

Unveiling of the Daniel Fiske Jones Memorial (Moseley Auditorium).

WEDNESDAY EVENING, OCTOBER 16
8:15

Sanders Theater Exercises, Harvard University (admission by invitation only).

Rt. Rev. Henry K. Sherrill, *Chairman*

The Need for Wider Research. Dr. Raymond B. Fosdick, New York. President, Rockefeller Foundation.

The Influence of the Discovery of Ether on the Development of Surgery. Dr. Evarts A. Graham, St. Louis. Bixby Professor of Surgery, Washington University School of Medicine, and surgeon-in-chief, Barnes and St. Louis Children's hospitals.

The Emergence of Anesthesia's Second Power. Dr. Henry K. Beecher, Boston. Henry I. Dorr Professor of Research in Anesthesia, Harvard Medical School, and chief of the Anesthesia Service, Massachusetts General Hospital.

Medical Interest in Radioactivity from Becquerel to Bikini. Dr. Karl T. Compton, Boston. President, Massachusetts Institute of Technology.